VOLUME I

CLINICAL TUBERCULOSIS

CLINICAL TUBERCULOSIS

BY

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WITH A CHAPTER ON LABORATORY METHODS

BY

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VOLUME I

PATHOLOGICAL ANATOMY, PATHOLOGICAL PHYSIOLOGY, DIAGNOSIS, AND PROGNOSIS

SECOND EDITION

WITH ONE HUNDRED AND FIVE TEXT ILLUSTRATIONS AND CHARTS, AND SIX PLATES IN COLORS

ST. LOUIS
C. V. MOSBY COMPANY
1922

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(Printed in U. S. A.)

Press of
The C. V. Mosby Company
St. Louis

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200
P851c
1922
v.1

TO

SIR JAMES MACKENZIE, LUDOLF KREHL, AND
GEORGE W. CRILE

Clinicians who Typify that Growing Spirit in Internal Medicine which
Recognizes the Importance of the Study of Pathological
Physiology and Emphasizes its Application,

THIS WORK IS DEDICATED

By the Author.

DISPENSARY, U. OF C.

707293

INTRODUCTION TO SECOND EDITION

It was with some misgivings that this work was first given to the public, for I realized that it was rather encyclopedic in scope and that it dealt with the subject in a manner, and from an angle, which had not been employed by other writers. I relied, however, on the fact that there was no work accessible to English reading clinicians which attempted more than a hurried discussion of the subject; and the searcher for the reason for things was usually disappointed in not being able to find it. It seemed to me that a fuller discussion of tuberculosis than that usually given was desirable; and that a consideration of the tuberculous patient and his treatment from a physiological standpoint was badly needed. Therefore, I hoped that a fairly complete discussion of tuberculosis and its problems might be welcomed. In this, I have not been disappointed, for the work has been accorded a very sympathetic reception.

In this second edition, I have attempted to keep abreast with our advancement. In the chapters on Diagnosis I have incorporated my latest observations on pulmonary reflexes and shown the path through which pulmonary tuberculosis expresses itself in disturbed function in the production of subjective and objective symptoms. The chapters on the Nervous System, in which are found the basic principles for understanding the manner in which the patient reacts toward tuberculosis in the production of symptoms, have been brought up to our present physiological knowledge; and, throughout, an effort has been made to still further emphasize and to more accurately classify the reactions shown on the part of the patient toward the disease. In harmony with the nomenclature adopted in my book on Symptoms of Visceral Disease, the term "parasympathetic" has been substituted throughout, for the term "greater vagus" which appeared in the first edition.

One new chapter has been added, "Influenza and Tuberculosis," which it is hoped will be appreciated by those who are experiencing the difficulties of diagnosis made by our recent pandemic. It is hoped that this second edition will be accorded the same generous reception as the first.

F. M. P.

INTRODUCTION TO FIRST EDITION

During recent years medical literature has abounded in papers and books treating of the subject, tuberculosis. Many of these contributions have been excellent. The majority of them, however, have been written from a viewpoint which is entirely too narrow. If we are to make advances in our knowledge of tuberculosis we must take a broader view than that expressed by the prevalent idea, that tuberculosis is a disease due to the tubercle bacillus, which produces a group of tubercles in the lung, and that its cure comes about as a result of good food and open air. We must look upon it as being an infectious disease producing inflammatory processes in some organ, or organs, of the body, but indirectly influencing every organ and cell of the body; and prior to the time that a specific cure has been found, we must look upon treatment as being the application of a sufficient number of remedies and measures to raise the patient's defensive powers sufficiently high to destroy the tubercle bacilli and to furnish the focal stimulation necessary to hasten scar formation. It is from this standpoint that I have approached my subject. Anatomy and physiology, both normal and pathological, have been made the basis of my studies; and visceral neurology has received unusual attention. I have endeavored to approach the study of tuberculosis from the standpoint of internal medicine in its broadest sense.

I am endeavoring to present in this monograph a record of the observations which I have made during twenty years of clinical study. The studies, for the most part, have been made on patients in the Pottenger Sanatorium for Diseases of the Lungs and Throat, Monrovia, California. My patients have represented all stages and phases of the disease. For the most part, they have been suffering from advanced widespread lesions. They have been of an unusually intelligent class, and this has added greatly to the opportunity for careful study.

In my discussion of the pathological problems I have endeavored to make the presentation as practicable as possible.

My viewpoint is that of a clinician. While I have in no manner ignored the subject of cellular pathology, I have borne in mind and discussed more extensively that side of pathology which is of greater importance to the clinician, namely, functional pathology. Scientific medicine is just beginning to appreciate the importance of this study. In the past, functional disorders were considered as being of little concern and not worthy of legitimate study; but, as our knowledge of visceral neurology and biochemistry increases, we, as clinicians, are learning that we should give as much, if not more attention to functional derangements than to actual organic diseases.

I have discussed the nervous system more extensively than is usual in a work of this kind, and have particularly emphasized the importance of understanding the relationship of the vegetative nervous system to disease. I have endeavored to indicate the important part that it plays both in being acted upon by the disease and its products, and in influencing the cellular activity in other organs and cells when so acted upon. While my discussion of this subject is necessarily more or less unsatisfactory because of the many gaps in our knowledge, yet I have endeavored to make my treatment sufficiently complete to give those who have not had their attention directed to the importance of visceral neurology a working basis for further study in this field of investigation.

The study of phthisiogenesis is discussed from the standpoint of tuberculous infection as a condition of childhood, from which clinical tuberculosis in the adult is a metastasis occurring after the early infection has changed the reactivity of the body cells and endowed them with the power of producing specific defensive ferments. The influence of this specific defense in warding off further infections and in changing the course of the disease by making it more chronic when implantation has occurred; likewise its influence upon the practical problems of prophylaxis, are carefully considered.

The subject of fever has received careful consideration and its relationship to the nervous system has been strongly emphasized. Anaphylaxis has been considered in its relationship to the parasympathetic division of the vegetative nervous system.

The power of the body to adjust itself to new and changed conditions has received careful attention. Such compensatory changes as occur between the two sides of the thorax; between the thorax and abdomen; and those which take place in the circulatory system, have received far more attention than is ordinarily given them.

The chapters on diagnosis, as well as those on therapy, are treated in their relationship to physiology and functional pathology.

The etiological classification of symptoms will be welcomed as an important advance which simplifies the picture of clinical tuberculosis and aids in its understanding.

Physical examination is considered in the light of my studies of the changes in the musculature and other superficial tissues as produced reflexly by the inflammatory processes in the lungs. The importance of inspection and palpation is emphasized; and these methods of examination, probably for the first time, are given their full value in diagnosis. Percussion and auscultation are described not only in relationship to the pathological process in the lung, but in relationship to all other changes in the organs within the thorax, as well as to the changes wrought in the muscles and subcutaneous tissues by the reflex motor and trophic impulses produced by the pulmonary inflammation. This new point of view offers an explanation of many of the errors which have always attended these methods of examination.

I have endeavored to look upon the therapy of tuberculosis from a common sense viewpoint. Realizing that the most important factors in the cure of tuberculosis are intelligent medical guidance and faithful co-operation of the patient, these points have been everywhere emphasized throughout this work. I have endeavored to explain the physiological basis of the action of the more important measures employed in treatment, in the hope that such an understanding will produce a more intelligent therapy than that which is practiced today. Not only have I pointed out the benefit to be derived from each of our more important measures; but, at the same time, have shown their limitations.

Psychotherapy is given an important place in therapeutics, and an attempt has been made to show the physiological basis of its action in the hope that it will find a wider application in practice.

The discussion of laboratory diagnosis is not intended to be a restatement of the ordinary routine methods which are employed in the examination of laboratory specimens, but a discussion looking towards more accurate methods and more valuable studies resulting from a critical study of laboratory specimens. This chapter is written by Doctor J. E. Pottenger, who has made the laboratory study of the patients in the Pottenger Sanatorium for the past eight years.

In the tuberculosis clinic I have endeavored to give to those who are interested, but less familiar with the course of chronic tuberculosis, a definite picture of this disease through a discussion of individual cases which have been under the writer's care. In this I endeavor to teach the early symptomatology; to show the mistakes in diagnosis; and, by following the case during its progress, to show the relationship between symptomatology and the pathological changes which are going on within the tuberculous process. While I realize that the temperature and pulse curves do not indicate fully the character of the pathological lesions within, yet I have used them to furnish a tangible measure of the toxemia present. When necessary, I add other important symptoms to make the picture clearer. I have attempted this in order to answer the many inquiries which I receive from men asking if such and such a case is not being harmed by some remedy or measure that is being used, or if some other remedy or measure will not benefit more. I wish to impress the fact that chronic tuberculosis is a disease which runs an uneven course and in which many periods of activity must be expected before a final result is attained; and show that these periods of activity are natural, and that they do not indicate that the patient is not improving satisfactorily.

The burden of my study in tuberculosis has been to find an explanation for the facts observed,—the reason why. I have made no effort to treat the subject in textbook style. The work should be looked upon as a series of monographs dealing with the subjects under discussion in a more or less complete manner. This has made a certain amount of repetition necessary, but, had it been omitted, it would have been to sacrifice the continuity of thought. Throughout I have endeavored to make my discussions practical. I have endeavored to correlate the pathology with the symptomatology, so that the reader will think of symptoms as being an expression of functional disturbance caused by pathological change; and have attempted to classify the symptoms so as to aid in locating the pathological process.

While much that I have written is incomplete, I trust that it will emphasize the importance of a study of this kind and stimulate others to pursue it further.

I have made no attempt to give an historical statement of the subject, nor have I attempted to review the excellent works of many observers. I have quoted freely, where the quotation added to my argument, or elucidated some point under discussion. The names of many excellent workers whose studies have added to the richness of our knowledge, and who have both consciously and unconsciously influenced my conclusions will not appear in the text, but I wish to acknowledge my indebtedness to them, the same as to those whose names are mentioned.

My thanks are due to many who have aided me in preparing this book, especially to Dr. J. E. Pottenger, for preparing the chapter on laboratory methods; to Dr. R. Walter Mills, for his paper on the relationship of physical types to the form and function of the internal viscera, and his excellent drawings to illustrate the same; to Dr. Albert Soiland, for making and reading the plates of the patients described in the chapter on x-ray diagnosis, and for furnishing the x-ray pictures for the cuts; to Drury Victor Haight, for preparing most of the illustrations and plates; to the Misses Paine and Hardy, for preparing most of the temperature charts; to Mr. Culver, for making the charts illustrating graphically the method of employing tuberculin according to the extent and degree of activity of the disease; to Mr. Lacy, for photographs and drawings; to M. S. Pottenger, for correcting and reading the manuscript; to Miss Henrickson and Mrs. Sanford, for copying the manuscript; and, last but not least, to my assistants and patients who helped me in making observations and collecting data.

I desire especially to thank the publishers for their untiring efforts to meet the difficult problems put up to them in the mechanical part of the work.

FRANCIS M. POTTENGER.

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VOL. I.
CLINICAL TUBERCULOSIS

CHAPTER I.

PATHOLOGICAL CHANGES IN TUBERCULOSIS.

The changes which take place in the tissues as a result of tuberculous infection are many, and vary according to the location and severity of the infection. Aside from the formation of the tubercle and the evolutionary changes through which it passes, there are changes in adjoining tissues which are due to the toxins or different bacillary substances which diffuse into them. There are also changes of a non-tuberculous nature such as the various degenerations, the interference with circulation resulting in congestion, the pathological changes due to reflex action, changes in the nerves, in the blood vessels, and in the blood itself. In fact, no tissue of the body escapes unchanged during a tuberculous process of severity and long standing.

Tubercle.—When tubercle bacilli gain entrance into the tissues they at once set up an irritation which causes an inflammatory hyperemia. There is a gathering of leucocytes, particularly lymphocytes, and the fixed cells in which the bacilli are found respond to the irritation and proliferate. This reaction results in the formation of the tubercle. Within the tubercle lie the tubercle bacilli. The periphery of the tubercle consists of a ring of round cells, particularly lymphocytes. Within this lies another ring of cells, larger than the leucocytes, containing one nucleus the so-called epithelioid cells from their resemblance to the epithelial cell. These cells are probably, although not definitely proved, derived from the fixed tissue cells, in which the bacilli are found. In the center of the tubercle may be found giant cells. These were formerly thought to be essential to the tubercle and characteristic of tuberculosis, but are now known to be sometimes wanting and sometimes found in other conditions. The giant cells when present form the center of the tubercle. They vary in size and shape and contain many nuclei. They are probably an expression of necrosis and disappear as the necrotic process extends, the new ones taking their place peripherally.

The growth of the tubercle is slow. In two weeks from the time infection occurs, it is but large enough to be detected microscopically. Fig. 1 is a schematic illustration of a tubercle.

When necrosis occurs and extends it does so by gradually involving the peripheral cells. If the infecting microorganisms are of low virulence or if the biochemical properties of the tissues are resistant, the disease takes upon itself a chronic course, and the tissues surrounding the tubercle form new connective tissue, which tends to wall in or to separate the focus from the adjoining structures. If the tubercle is small it may be changed into connective tissue and leave no visible marks of its existence. If larger it leaves connective tissue in its place. Fig. 2 illustrates the conversion of tubercle into fibrous tissue.

At the same time that the leucocytes are gathering and the epithelioid cells are forming in order to surround the bacilli, and guard the organism from their further attacks, the bacilli are multiplying and preparing to wage an offensive campaign. Activity in a tuberculous process means that the bacilli are multiplying. Bacillary products stimulate the body cells, which in turn produce ferments which destroy the bacilli. Whether necrosis results from a ferment produced by the bacilli or is due to the exhaustion of antiferment in the focus is not clear. Fig. 3 illustrates a tubercle with necrotic center.

Collateral Inflammation.—Aside from the pathological changes which are caused directly by the bacillary process there is an inflammation of greater or lesser extent and severity affecting the tissues adjacent to the tubercles. This collateral inflammation depends upon the virulence and number of bacilli on the one hand and the receptivity of the tissues on the other. It may be a productive inflammation or a degenerative process. The exudation may be serous, fibrinous, cellular, sanguinous, or purulent. It may undergo the same changes as the tubercle itself, producing new connective tissue, or softening. Formerly it was thought that these changes were due to other bacteria, so-called mixed infections; but now it is known that all these changes can be produced without the help of any other organism than the tubercle bacillus.

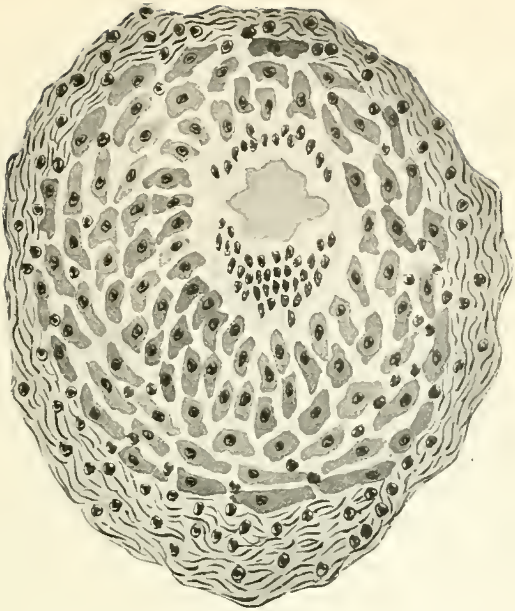

Fig. 1.—Schematic illustration of a tubercle. Center, necrotic giant cell; second layer, epithelioid cells; outer layer, lymphocytes. (Adami.)

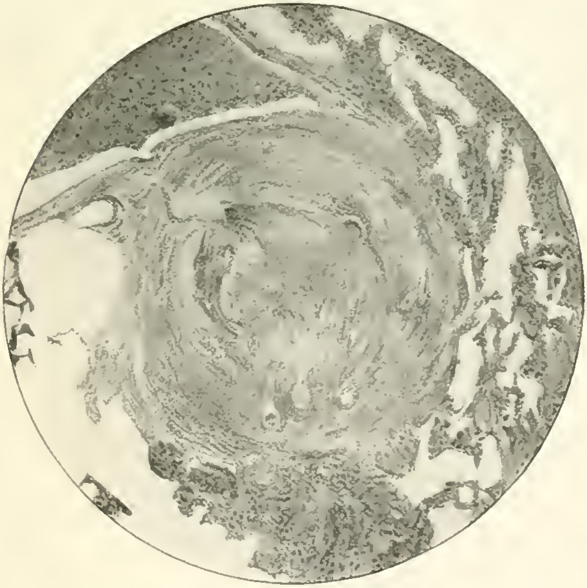

Fig. 2.—Illustrating conversion of tubercle into scar tissue. Tubercle undergoing fibrosis; center partially fibrous. (Fowler and Godley.)

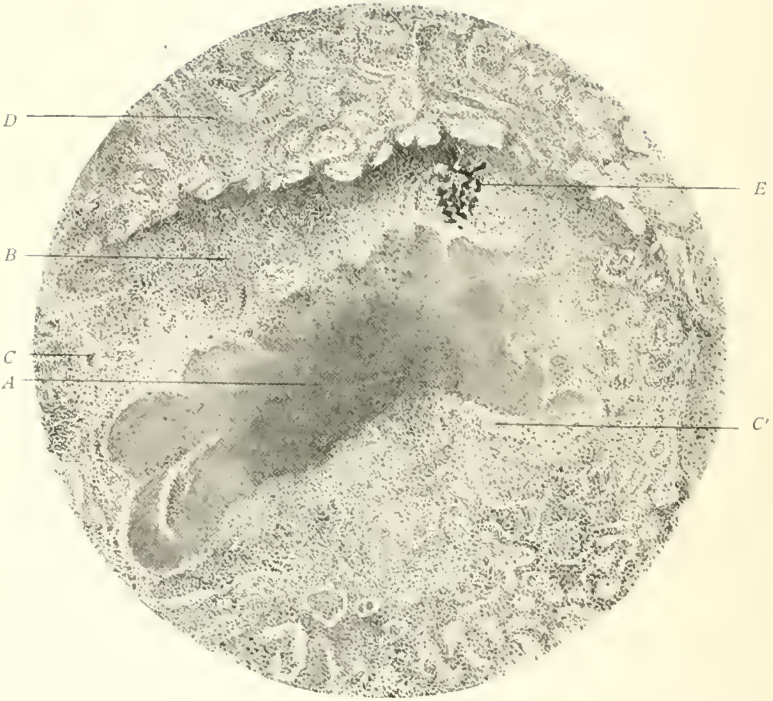

Fig. 3.—Illustrating conversion of tubercle into necrotic tissue. Tubercle undergoing caseation. *A*, caseous central area; *B*, cellular peripheral area; *C-C'*, giant cells; *D*, small-celled infiltration of surrounding alveolar walls and spaces; *E*, pigment. (Fowler and Godley.)

The collateral inflammation is due to substances which, passing out from the tubercle, diffuse into the tissues. These may be specific substances, produced during the growth of bacilli, or substances of a fatty or albuminous nature which are derived from the destruction of the body of the bacillus. A large factor in the production of the inflammation is the presence of proteolytic enzymes. The inflammation is most severe near the tubercle and less so further away.

Bacilli often pass from within the tubercles out into the tissues affected by collateral inflammation, and set up new foci of infection. These metastatic foci are more serious than the collateral inflammation alone. The tissues which are affected by the collateral inflammation are especially prone to infection because the cells are injured by the exudative process and there is a stagnation of blood and lymph which prevents the free access of antibodies; and further lodgment of bacilli in the tissues is favored by the lessened motion of the part.

Recognition of Tuberculous Inflammations.—Tuberculosis shows every form of inflammatory change; and while there are certain changes which are most often found in tuberculous tissue, yet there are none that are absolutely characteristic. Exudates in which lymphocytes predominate are considered to be of tuberculous nature, while those in which polynuclears predominate are considered non-tuberculous. This will not always hold true. Polynuclear leucocytes sometimes predominate in the cerebrospinal fluid in tuberculous meningitis in the early stage of the disease; and lymphocytes may be found in large numbers in any chronic non-purulent exudate. Necrosis and cheesy degeneration are found in other inflammations as well as tuberculosis. Even the tubercle, as previously mentioned, with its giant cells may be found in other inflammations.

Conglomerate Tubercle.—Large foci are often formed by a number of tubercles fusing together. Such conglomerate tubercles, as they are called, may be only as large as a small marble, or they may be as large as one's fist.

Tuberculous Ulcer.—One of the common results of the tuberculous inflammation is the tuberculous ulcer. This is a result of the degenerative changes in the tubercle; or, if large, in the conglomerate tubercle. As long as tubercle bacilli are capable of multiplying they give off toxic substances; and both during growth and after destruction yield substances belonging to the fatty and protein groups, which produce necrosis of the cells with which they come in contact. This softening of the tuberculous mass is usually accompanied by an inflammatory process and a rupture of the walls confining it. If it is somewhere on the surface, such as in the larynx, or pharynx, a superficial ulcer remains. If it is deep in the tissues, as in the lung, bone, or kidney, a rupture is followed by a loss of tissue, a cavity remaining.

All tuberculous ulcers and cavities are formed in the same way, first by a deposit of bacilli; second, a softening of a mass of cells with a separation of the tissues, then a rupture, usually near the center. As a result it can be seen that the edges of a tuberculous ulcer are undermined. This is illustrated schematically in Fig. 4, in which *A* represents implantation of bacilli and induration of tissue; *B*, softening of center; *C*, rupture and expulsion of abscess contents with edges of surrounding tissue projecting beyond the abscess walls.

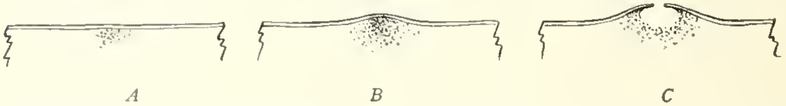

Fig. 4.—Schematic illustration of the manner in which tuberculous ulcerations or cavities are formed. *A*, deposit of bacilli; *B*, necrosis of the mass; *C*, the rupture with expulsion of the contents.

A tuberculous cavity may be one single abscess cavity with regularly formed walls, or it may be irregular, the result of many adjoining abscesses. The walls may be smooth and regular, or irregular. They may always secrete or they may eventually dry out. They may always remain as open cavities or they may, especially when small, be compressed as contraction occurs, and be either partially or wholly closed. The walls of cavities when chronic, may be made up of fibrous tissue one or more centimeters in thickness.

Cavities are often a source of bleeding. Sometimes the blood is scant, and is thoroughly mixed with pus from the cavity, giving a pinkish appearance. This is, as a rule, from delicate new blood vessels. The most serious hemorrhages, however, often come from larger vessels in cavities. Where cavities form, the vessels, as a rule, are closed off by inflammatory changes in their walls, but now and then either the vessel is too large or the destructive process in the tissues comes too rapidly and the contents are evacuated while the vessel still remains patulous. The supporting tissue being taken away from the walls of the vessel, it dilates, forms an aneurism, becomes thinner and thinner, and eventually ruptures. In this manner many fatal hemorrhages occur. This is illustrated in a schematic way by Fig. 5.

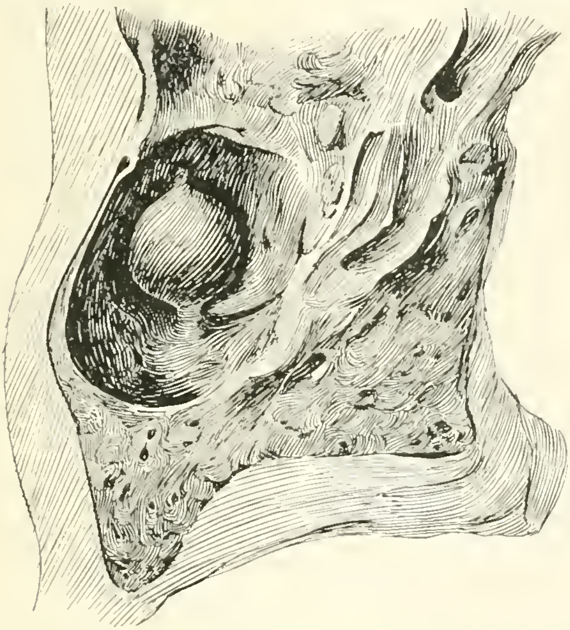

Fig. 5.—Illustrating schematically aneurismal dilatation of a vessel in a cavity, rupture of which often produces hemorrhage.

Avenues of Infection.—There has been much discussion during recent years as to the modes of entry of the tubercle bacillus into the human body. Formerly it was taken for granted that it must be through the air passages because the lungs were usually involved. Later, entrance through the alimentary tract was proved, and this assumed a prominence which, for a time, completely overshadowed the route of the air passages. Animal experiments have been performed that show that both routes are capable of carrying the bacilli to a safe lodgment in the tissues. They also enter through the skin, but this route is of comparatively little importance.

Bacilli do not require injured mucous membranes to facilitate their passage, but penetrate through the intact surfaces. Bacilli pass through the intestinal wall and may be carried to the liver, or lymphatic glands; or, through the thoracic duct and pass directly into the blood stream. Bacilli injected into the rectum have been found within a few days in the mediastinal glands and lungs. Bacilli seem to find their way to the peribronchial glands no matter how they enter the body. Until this point was proved it was thought that infection must be nearly all aerogenous because of the frequent involvement of the lungs and bronchial glands.

In 1912 Ghon[1] published the results of a painstaking postmortem examination of chests of children having enlarged peribronchial glands and showed in every case where he found certain peribronchial glands infected that, by examining that portion of the lungs, whose lymph drained into these glands, he could demonstrate a tuberculous nodule. This work seems to substantiate the aerogenous theory; but when we recall that bacilli might be taken through the walls of the alimentary canal and be carried through the thoracic duct and poured into the blood which passes through the lung before passing on to the general circulation, we can see that even alimentary infection might produce foci in the lungs without primary involvement of the lymphatic glands.

The route of entry is of practical importance in the solution of the question of the relative danger of bovine and human infection. The chief source of infection from cattle is milk; and the chief avenue of infection is through the alimentary tract. On the other hand, bacilli of human origin may be found in the air we breathe, especially in closed rooms occupied by open cases of tuberculosis living under unfavorable hygienic conditions; in food we eat; or in droplets of sputum thrown from the mouth of the coughing patient (Flügge). So, human bacilli may be taken into the body either through the air passages or the alimentary tract. No matter in which way they enter, the process is the same.

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[1] Der Primäre Lungenherd bei der Tuberkulose der Kinder, Wien, 1912.

While it is generally believed that nearly all cases of pulmonary tuberculosis in the human being are due to human tubercle bacilli, and that the extrapulmonary lesions are more apt to be due to bovine bacilli; this question is far from being proved, and cannot be decided on the basis of the routes of infection. Some have thought that the adaptability of the tissues to the particular strain might be a factor in localization, while the bacilli of both varieties gain entrance through the same avenues. Both types have equal advantage in gaining access to the lymphatic system and probably follow much the same laws in their spread. Neither can it be determined by the severity of the process; for, as a general rule, the method of producing metastatic infection, has much to do with the virulence of the process. Processes which result from blood metastases, such as the early lesions in the lung, those in bones, joints and the kidneys are mild infections, while those which spread through the lymph channels, such as the secondary metastases in tissues adjacent to other foci, and those which spread through the bronchi are apt to be serious infections. For a more complete discussion see page 57.

The Localization of Tuberculosis.—No matter whether the bacilli gain entrance through the air passages or the gastrointestinal tract their localization and further spreading in the body seems to take much the same course. Whether the bacilli gain entrance through the one tract or the other, they usually, eventually, in some manner, make their way to the lymphatic glands and very frequently to the peribronchial and peritracheal glands. The mesenteric glands may be infected when bacilli enter through the gastrointestinal tract, the same as the cervical when the tonsil is the point of entry; but, on the other hand, it is not necessary that such should be the case, for bacilli may be carried from the tonsils, intestinal walls or other mucous surfaces to the lungs and mediastinal glands, leaving these regional glands in a healthy condition. The passage of bacilli through groups of glands is particularly apt to occur in childhood because of the peculiar characteristics of the glandular structure in early life. Bartel[2] shows that the canals in the lymphatic gland of the child are wide, while those of the adult are narrow, thus permitting bacilli to pass on in the former, while they are detained in the latter. In the former bacilli may pass on through several glands before finding lodgment. Lack of specific cellular defense must also be a factor in the case of penetration.

Even if the primary extraglandular focus should be somewhere in the lungs, as has been shown by Ghon, the particular bronchial glands which drain the area also become infected, enlarge and remain a nidus whence further metastases may occur. The primary focus is, as a rule, small. The glandular focus, on the other hand, even though secondary, is apt to be large and out of all proportion to the primary lesion. This is illustrated in Fig. 6, from Ghon's monograph. See further discussion in Chapter II.

From the fact that the glands bear such a prominent part in early infections, tuberculosis must be looked upon as being primarily a disease of the lymphatic system which produces secondary or metastatic infections in almost any organ of the body, but which shows a marked predilection for the lungs. It might be suggested that one reason why the lungs are so often the seat of involvement in metastatic tuberculosis is because the bacilli pass from the infected lymphatic glands into the blood vessels and are carried through the lesser circulatory system to the lungs before circulating in other parts of the body. But this theory leaves some phenomena still unexplained, especially since we know the bacilli are often found in the systemic circulation of patients suffering from tuberculosis (Rosenberg), particularly advanced tuberculosis. We know no reason why we should not assume that the pulmonary tissue is particularly favorable to the growth of the tubercle bacillus the same as the tonsil is favorable to the diphtheria bacillus.

Rosenow has shown that streptococci cultivated from the thyroid localize preferably in the thyroid in experimental infection; those grown in the tonsil, in the tonsil; and the same for bacteria from other tissues. It is quite reasonable to suppose that the same law operates in the localization of the tubercle

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[2] Der normale und abnormale Bau des Lymphatischen Systems und siene Beziehungen zur Tuberkulose; Festschrift zur VI Internationalen Tuberkulosen Konferenz, Wien, 1907.

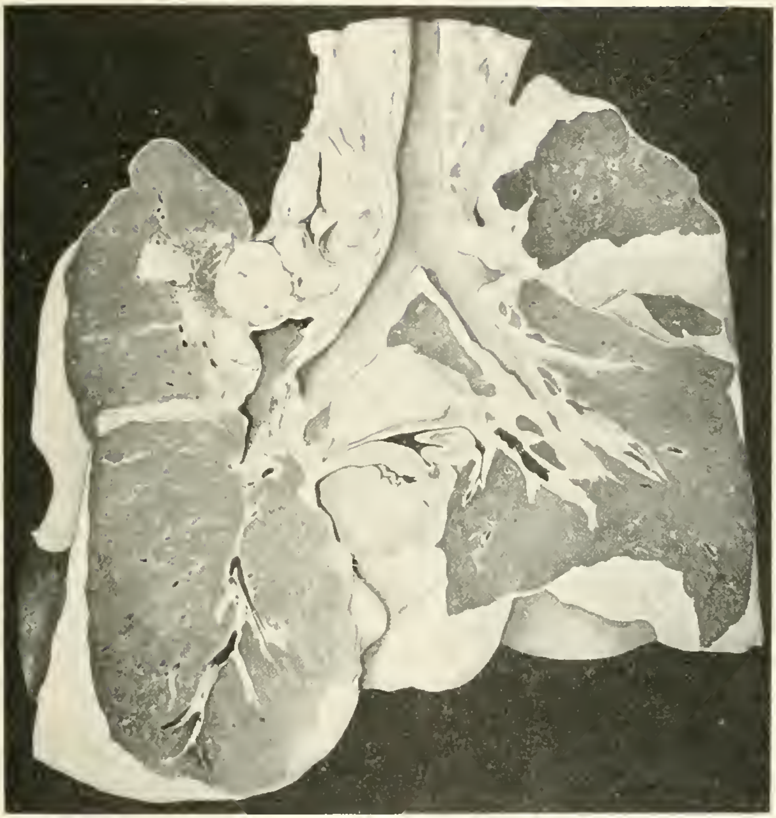

Fig. 6.—Illustrating disproportion between lesion in the lung and the enlargement of the peribronchial glands in a child in whom immunity has not been established by previous infection. Primary caseous focus in the left upper lobe, with miliary tubercles in the surrounding area. Caseation of the bronchopulmonary lymphatic glands adjoining the left upper lobe, alongside of the partially visible dissected bronchus. Caseation of the upper tracheobronchial lymphatic glands on the left, and adhesion of these lymphatic glands with the medial surface of the left upper lobe. Acute miliary tubercles in the lower tracheobronchial lymphatic glands. Scattered acute miliary tubercles in both lungs. The upper tracheobronchial and bronchopulmonary lymphatic glands on the right are unaltered. The preparation was preserved in accordance with Kaiserling's method, and is taken from a four-year-old boy. Reduced photograph of the preparation. Posterior view. (Ghon.)

bacillus. It is in full harmony with our ideas that bacilli grown on a given medium assume peculiar characteristics and become more and more partial to that medium.

We must still account for the fact that the apex is most often the seat of infection. This question is discussed in Chapter V, and needs no further discussion at this time.

Differences in Air-borne and Blood-borne Intrathoracic Diseases.—Bearing upon the question of the route of infection and the special predisposition of the apex to infection in tuberculosis, important information can be gained by studying other infections of the lower respiratory tract. The special bacteria which produce the various pneumonias and infections of the bronchial tract are air-borne and often found in a saprophytic state in the air passages, particularly those of the upper respiratory tract. These diseases may be localized in any portion of the lungs, but are particularly prone to infect those portions of the lung which receive the air currents most directly, and in which respiratory movement is greatest; consequently the pneumonias (lobar) most often affect the lower lobes; and, where other lobes are also involved, the disease usually makes its first appearance in the lower lobe. Bronchitis and bronchial pneumonia may affect any portion of the lung or lungs; but in bronchitis, as a rule, the infection begins in the trachea and large bronchi where the parts are most exposed to the air currents and extends progressively toward the fine ramifications of the air passages. If the smaller tubes and air cells become involved, causing a bronchopneumonia, it usually shows itself first at the bases, or, at least, over the lower portions of the lungs. Thus the common, definitely accepted air-borne diseases of the lungs affect the larger bronchi and the lower lobes, where the entrance of the air is most direct and the movements greatest; and we would expect tuberculosis, if it were an air-borne disease, to have the same localization. On the contrary, disregarding lesions which spread contiguously from the hilus, the pulmonary involvement in tuberculosis, as we find it clinically and pathologically, usually affects first the apices of the lungs and then spreads downward involving contiguous or nearby tissue. This indicates a distinct difference in the mode of infection for bronchitis and pneumonia on the one hand and tuberculosis on the other; and since we know that bacilli are carried to the lung, both by the blood and lymph streams, also along the paths of the bronchi in secondary metastases; and, since we also know that a slowing of the blood and lymph current takes place at the apices, favoring the deposit of bacilli in both blood and lymph channels, we are justified in suspecting the tubercle bacillus to follow one or both of these modes of gaining access to the pulmonary parenchyma. This indicates that pulmonary tuberculosis is metastatic tuberculosis, secondary to other foci. Since there is a wide gap between the primary focus in the gland (usually in the tracheal and peribronchial group) and the apex of the lung where the first pulmonary metastatic focus occurs, we must assume that it is far more probable that the metastasis is a result of infection in the direction of the natural currents of the blood than that it should take place against the current of lymph which flows from the apex toward the hilus.

The Effect of Cell Sensitization Upon the Implantation of Bacilli.—We cannot consider the train of events which takes place in the spreading of tuberculosis from the lymphatic system to other tissues, without at the same time considering the degree of immunity present in the patient's body and the immunity reactions which occur between the tissues and the bacilli which have made their escape from the previous foci.

We are probably justified in assuming, if we interpret experimental results correctly, that every patient who has an infection of any portion of the body has struggled with the bacillus; and while it may be that he has not been fully able to overcome it, yet he has at least developed a more or less marked immunity against it. And we must also believe that a more or less continuous inoculation, with its resultant immunization, goes on from this glandular focus until in favorable cases the bacilli cease to multiply and the focus heals; and in less favorable conditions, although healing may not occur, yet the multiplication of bacilli is usually checked and the activity of the disease process held in abeyance for a time.

The child, coming in contact with tubercle bacilli, usually meets them at first in small numbers, and is able to overcome them; or, at least to confine the infection to one of mild degree. Where an infection has resulted and been confined to a small focus, whether this heals or not, the organism develops such a degree of immunity, that is, the cells develop such a power of specific defense that it is able to defend itself almost wholly from further infection from without.[3] Koch's original experiment showing that whereas a healthy guinea pig infected with tubercle bacilli, shows a ready absorption of the bacilli and an infection of the regional lymph glands, while one which was previously tuberculous, shows a resistance to infection, a local ulcer forming, the bacilli being expelled and the regional lymph glands remaining unaffected, confirms this.

Secondary, or metastatic infections, such as those affecting the lungs, are for the most part believed to be due to bacilli escaping from some previous, usually lymphatic, focus. Here, too, we must suppose that the number of bacilli escaping is usually small, and that most of them are destroyed without an infection occurring. The probabilities are that if they are not destroyed while coursing in the blood stream, they are reduced in virulence. Many of them, we assume, are destroyed as a result of their stimulation of the sensitized cells to the production of specific protective ferments, as soon as they are enmeshed in the pulmonary tissues. However, now and then, regardless of the specific cellular defense (immunity) present, an infection will occur. This metastatic focus, as a rule, is small and single; the exception being when a large number of bacilli have made their escape from the lymphatic focus, such as occurs when softening of a large tubercle takes place with rupture into a blood vessel, producing either a localized or a general dissemination (miliary tuberculosis).

The body fluids containing antibodies, the pulmonary cells being specifically sensitized and the number of invading bacilli being small, the early metastases, as a rule, do not make rapid progress. They are fibroid in form. They often amount to no more than a nodule which can be found only by most careful search; at times, however, the infection is more widespread and yet results in new tissue formation. Occasionally, however, the primary metastasis will be transformed into an area of necrosis, either small or large, which may either become calcified and remain quiescent, or rupture, leaving an open ulcerating surface, this again to heal or remain as a focus from which further metastases may form.

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[3] Romer: Brauer's Beiträge zur Tuberkulose, Bd. xiii, xvii, and xxii.

These primary foci in the lung often become encapsulated with bacilli enclosed; and, while the bacilli may go on to death, yet at times they remain alive and sooner or later, for some cause which we do not understand, begin to multiply and produce activity in the focus of infection. Von Pirquet has assumed that at times the body takes upon itself a condition wherein, temporarily, it loses its specific defense against bacilli, and in this way he accounts for the renewed activity in old quiescent foci. This condition has been termed "anergie" by him. Any focus which has not thoroughly healed with destruction of the bacilli, may be a source of further dissemination of the disease.

From these primary metastatic foci the disease may spread at once or later, either by way of adjacent lymph spaces or small bronchi; pushing out into the adjoining tissues and gradually involving more and more of the lung. The extension usually takes place downward from the apex. Should the bacilli gain access to the blood stream, metastasis may occur in distant portions of the lungs or even distant parts of the body. But the general rule is for tuberculosis to extend contiguously.

The first pulmonary metastasis of hemotogenous origin usually occurs near the apex, nearly always above the third rib. This is the same area that usually shows infection of the pleura. Aside from these areas there is often a primary infection of those pulmonary and pleural tissues which are contiguous to the bronchial glands, the infection apparently extending against the lymph stream into the tissue.

Lymphogenous Metastasis.—The spreading of pulmonary tuberculosis through the lymphatics is of special import. It is probable that bacilli are carried by the wandering cells. Some authors attribute great importance to this method of spread. Again, it may spread through the lymph spaces. In this connection it is necessary to understand the influence of respiration in both the healthy and diseased lung. Every inspiration increases the pulmonary area and widens the intrapulmonary lymph spaces; every expiration lessens the pulmonary area and narrows the lymph spaces. By this succession of enlarging and narrowing the lymph spaces, the fluid is moved back and forth through the pulmonary tissues. Not only is the lymph forced toward the larger lymphatic trunks and regional glands, but it can easily be seen that it might be forced backwards as well. This occurs often; and it is now generally conceded that bacilli may be forced onward in the general direction of the lymph flow or backward against the stream (Tendeloo). Another point of great interest is that there is a wide difference in the lymph flow in various portions of the lungs, also in the healthy as compared with the tuberculous lung. The motion of the chest wall and diaphragm is lessened on the affected side. The respiratory effort is restricted and consequently the flow of lymph is retarded throughout the organ, a condition which favors the implantation of bacilli in the lymph spaces.

Lymphatic metastases are often found in the glands of the lung, the lymph spaces surrounding the bronchi and vessels and the subpleural spaces.

The fact that tubercle bacilli may be carried against the lymph stream assumes special importance in connection with the theory of certain authors that infection travels from the hilus toward the apex.

The conditions which favor lymphatic metastases are the collateral inflammation and the restricted motion of the part. These are accompanied by injury of the tissue and a retarded lymph flow. If only a few bacilli escape into the adjacent lymph spaces the resulting infection may be mild, but, if the numbers are great the best conditions possible are present for an extremely virulent metastasis.

Bronchogenous Metastasis.—While the lessened motion of the diseased lung together with the stasis dependent upon the collateral inflammation favor lymphatic spread of the disease, it lessens the danger of spread by the bronchi. This latter is a very common form of spreading of the disease in open cases. When the disease has passed to the point of softening and cavity formation, the air passages are always exposed to the danger of infection from this source. Deep breathing and coughing has a tendency to force the bacillus-bearing sputum into areas not already infected, to plug the bronchus with infectious mucus, thus starting up new foci of infection. When bacilli are implanted upon the tissues through the bronchial route, they do not affect the epithelium of the air passages, but pass through the mucous membrane and settle in the subepithelial cells. This type of the disease is apt to be very virulent. This is the usual method of infection in circumscribed caseous pneumonias.

Hematogenous Metastasis.—Hematogenous metastasis shows many variations. It has been proved recently that tuberculosis is at times a bacillemia. Bacilli often pass into the blood stream; and they can be recovered in a large per cent of patients suffering from the advanced form of the disease. When only a few bacilli enter the blood stream, they are destroyed without an infection resulting; when the number is larger they may withstand the effects of the antibacillary elements in the blood, likewise the specific defensive properties of the cells in which implantation occurs and form a new focus; when still larger the bacilli may be in such numbers, or when clumped, they may heap up, blocking the capillaries of either a particular, limited area supplied by a certain small blood vessel and its branches or they may scatter through a large area of the body, causing acute general miliary tuberculosis.

The hematogenous form of spreading accounts for the infection of an organ distant from the primary focus. Thus, such lesions as those of the bones, joints, meninges, and kidneys occur when the primary focus is in the mediastinal glands or lung.

Ordinarily, except in acute miliary tuberculosis, the lesions which are caused by spreading through the blood are of a mild, non-virulent type. This is probably due to the fact that bacilli, entering the blood stream, come in contact with the specific antibacillary substances so intimately that while not wholly destroyed, their virulence is greatly reduced, and, further, they are so scattered by the flowing blood that the infection which occurs is formed by few bacilli. Widespread fibroid tuberculosis is produced in this same manner.

There has been much speculation on the part of students of tuberculosis in trying to explain why the areas above the third rib, and particularly those toward the vertebræ, are more prone to infection than other portions of the lung. The plausible explanation is that these areas naturally have less motion than other parts of the lung, consequently, have a comparative retarding effect on the blood and lymph flow which, in turn, favors the

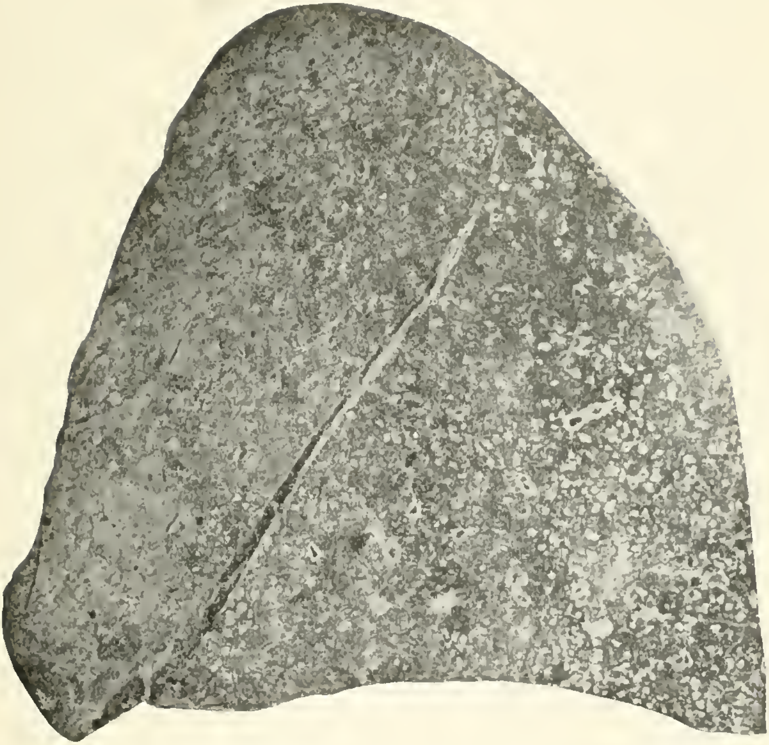

Fig. 7.—Miliary tuberculosis of the lung. (Tendeloo.)

heaping up of bacilli in the capillaries and lymph spaces, thus favoring infection. If, however, it were the lessened motion alone which favors infection, the disease would be most common at or near the hilus for it is at that point that the lung shows the least motion; but implantation is not favored at this point, either by way of the lymph or blood because the lymphatic trunks are larger and the capillary network is not as extensive as in the apex. It does occur, however, at times, as is shown by x-ray examination, but rarely causes clinical disease.

Freund, Schmorl and Rothschild have found certain conditions about the upper aperture of the thorax which they have described as being causative factors in the production of tuberculosis. Freund has found a shortening of the first rib and ossification of the first costal cartilage in many cases of tuberculosis and believes that these conditions act mechanically. This has been strongly supported by Hart. Schmorl has described furrows near the apex which he believes retard the flow of lymph and blood. Rothschild has found that there is a diminution of action in the manubrio-sternal joint in tuberculosis and described it as a predisposing cause. These theories fail to take certain facts into consideration. If the compression of the rib, or the furrow, or the lack of motion of the manubrio-sternal joint were the particular predisposing causes of underlying tuberculosis of the apex, we would expect the disease to begin in that portion of the lung most affected by such compression, namely, the anterior surface; but such is not the case. The disease usually begins posteriorly and near the vertebral column. Aside from this failure to conform to the facts of the localization of the disease, a more plausible explanation can be offered depending upon the anatomical differences incident to development and growth. In this connection I would refer the reader to Chapter V which treats of this subject more fully.

Forms of Tuberculosis.—Tuberculosis presents many different clinical pictures but there is no particular advantage in treating each as a separate entity. Pathologically we may classify the disease into those forms which have a tendency to produce new tissue, those in which destruction of tissue is the predominant factor, and the combination of these two.

Miliary Tuberculosis.—Miliary tuberculosis (Fig. 7), as previously mentioned, is caused by bacilli being thrown into the blood or lymph stream in large numbers. It is characterized by new tissue formation. There may be a large or small area involved. The tubercles throughout the area infected are of the same age and size having come from the same inoculation. Miliary tuberculosis may affect any organ. We see it in the lungs, spleen, liver, kidney, meninges, and peritoneum. If extensive, or if it affects a vital organ it is usually fatal in a short time. It may be generalized and affect many organs. One patient under my care, with miliary disease of the lungs, died on the twenty-first day after the onset of symptoms. Miliary disease of the lung often produces death before the tubercles soften. It may be a primary metastatic disease coming from some caseous focus in a patient who has been previously healthy, and apparently free from disease; or it may follow the breaking of a necrotic mass into the blood stream in chronic tuberculosis. I have seen it follow hemorrhages in a number of instances, sufficient bacilli gaining access to the blood stream to cause a generalized miliary condition and a rapidly fatal issue.

Fibroid Tuberculosis.—Fibroid tuberculosis is another form of the disease characterized pathologically by the formation of new tissue. This disease is usually chronic in character being produced by bacilli of low virulence. It is of lymphatic or hematogenous origin and the low virulence may be due to the fact that the bacilli are diluted, that their virulence has been reduced by the antibodies with which they have come in contact in the body fluids and the fact that the cells are endowed with the property of producing specific defensive enzymes before inoculation has occurred, as previously mentioned. The primary metastatic focus in nearly all organs is small in extent and fibroid in character, regardless of the form which the disease may assume later.

The virulence of the infecting bacilli in fibroid tuberculosis is so low that a very small amount of toxins are thrown out, consequently the tissues are irritated but not destroyed. This type is accompanied by a very mild collateral inflammation. The production of new granulation tissue results (Fig. 8). Sometimes this is very rich in blood vessels and results in large bundles of new tissue which later on are converted into hard

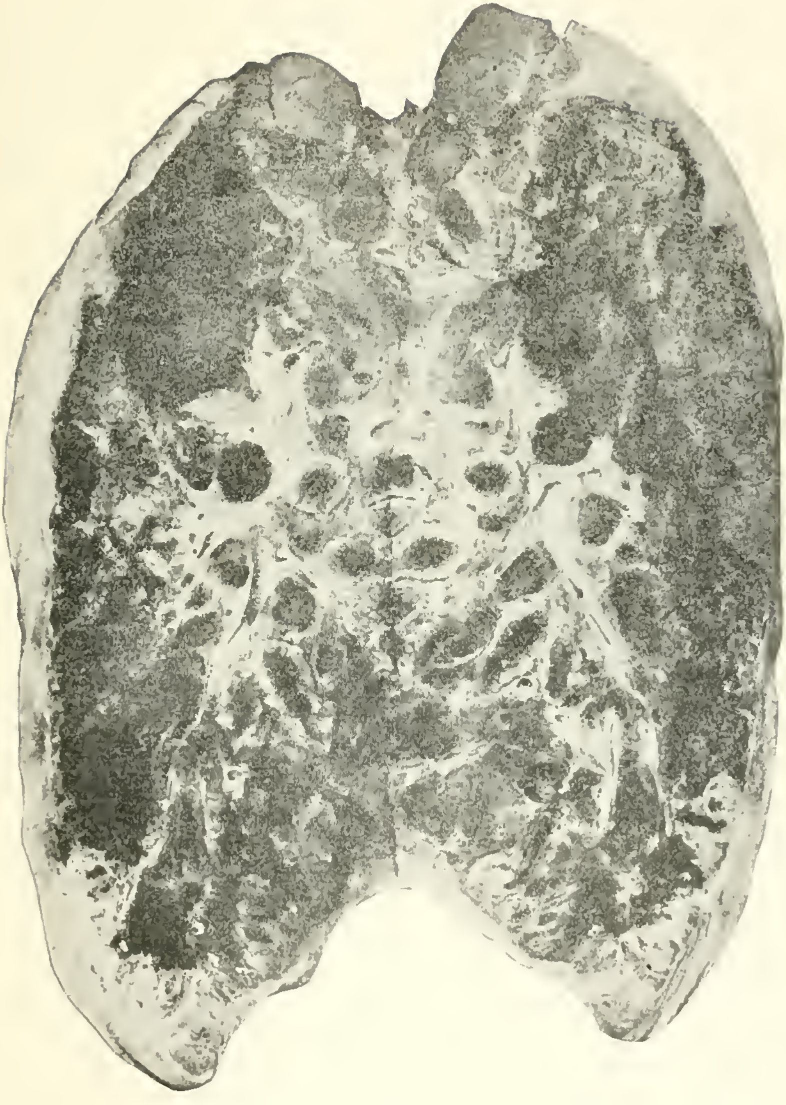

Fig. 8.—Illustrating pulmonary tuberculosis, with thickened pleura, many bronchiectatic cavities, and generalized cavity formation. (Tendeloo.)

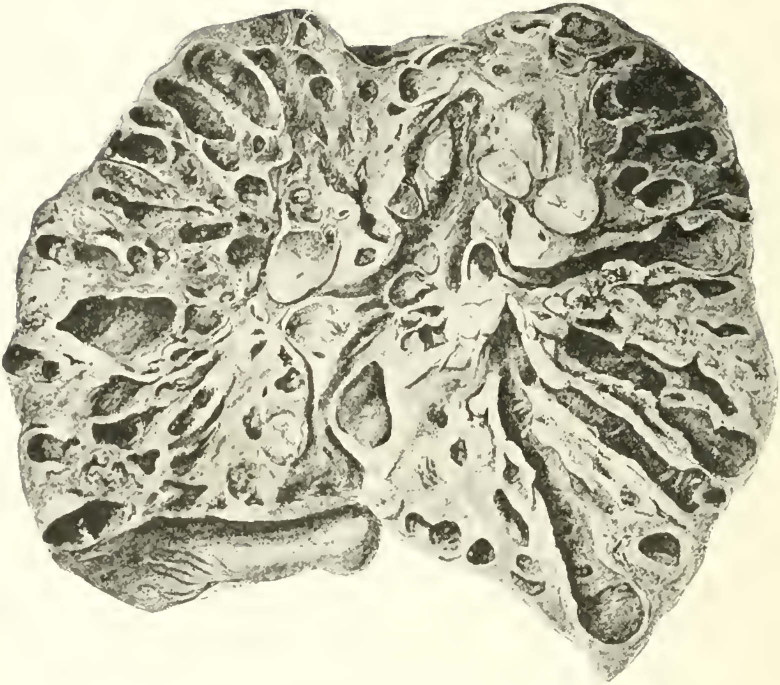

Fig. 9.—Sacculated bronchiectasis. (Powell and Hartley.)

scar which contracts and if extensive causes a great deformity of the organ. Resulting from the extensive contraction the lung becomes smaller. In order to compensate for the loss on the part of the affected lung, the other lung and the portions of the same lung which are not affected by the fibrosis, become emphysematous, the mediastinum draws toward the contracted side and all the thoracic organs are compelled to adjust themselves to new conditions, as described fully in Chapter XI.

Pouching of the bronchi may take place as a result of the irregular contraction causing a condition of bronchiectasis (Figs. 8 and 9).

This form of tuberculosis is accompanied by extensive connective tissue formation and the bacilli are well walled in. Many of them die. Others remain living but are rendered harmless. After a time, for some unexplainable reason, bacilli which have been shut up in the scar tissue and which have remained in a state of inactivity may develop an increased virulence. They multiply, produce toxins which are followed by necrosis and the conversion of the process into an ulcerative one. This is the course which many of the fibroid lungs eventually follow.

Caseous Tuberculosis.—Caseous tuberculosis (Fig. 10), is that form of the disease in which degenerative processes predominate. This form presupposes a virulent strain of bacilli and biochemical conditions of the tissues favorable to their rapid multiplication. These conditions result in a rapid development of toxins, with extensive necrosis, and a wide spread severe collateral inflammation. Acute tuberculosis, the so-called "galloping consumption," and acute pneumonic tuberculosis are of this type. These forms of the disease are characterized pathologically by rapid destruction of tissue with cavity formation if the patient lives long enough, and little or no tendency to connective tissue formation. If the process is extensive, marked toxemia and such a state of malnutrition accompanied by degenerative changes in the various organs occurs that the patient soon succumbs. If, on the other hand, the process is confined to a lobe or a portion of a lobe, the necrotic tissues may be expelled, a cavity resulting with relief of the toxemia and improvement of the patient. This is not an uncommon result.

Fibro-Caseous Tuberculosis.—This is the most common type of pulmonary tuberculosis met clinically. It is the usual form of chronic clinical tuberculosis. It consists of areas of necrosis which rupture and are thrown off leaving cavities; a collateral inflammation of a moderate degree; and a tendency to the production of new tissue in the areas which are the seat of tubercles which have not yet gone on to necrosis and softening; and further, a tendency to the formation of connective tissue about the walls of the cavities. In some patients the necrosis and ulcerative process predominates; in others the fibrosis. The process is chronic, and when progressive, gradually spreads from the apex downward toward the bases of the lungs. The greatest number of cavities is usually near the apex. Another favored seat is the apex of the lower lobe, although any portion of the lungs may be the seat of cavity. Cavities at the apices of the lungs are much more favorable for healing than those in the lower lobes because they are kept clean with greater ease, drainage being in their favor. The prognosis of this type depends much on the extent of the lesion and as to whether ulceration or fibrosis predominates. Sometimes an ulcerative process will predominate at first and fibrosis later, which usually means a retardation or an arrestment of the process; sometimes the reverse is true, which means progression and ultimate loss of life.

This form of tuberculosis, if an arrestment occurs, often shows even more marked contraction and compensatory changes with greater shifting of the mediastinum and diaphragm than the more purely fibroid form.

Non-tuberculous Changes in Other Organs.—In chronic pulmonary tuberculosis there are changes in the various organs of a non-tuberculous character which prove to be extremely important from the standpoint of prognosis, and which must not be neglected in therapy.

Pulmonary tuberculosis, after it has reached the chronic stage (consumption) cannot be looked upon as merely a disease of the lungs. It is a disease of the lungs caused by the implantation of the tubercle bacillus, plus all secondary changes resulting from and accompanying such infection; plus all the disturbances of function on the part of the other organs of the body which result from the changes in the lungs and from the

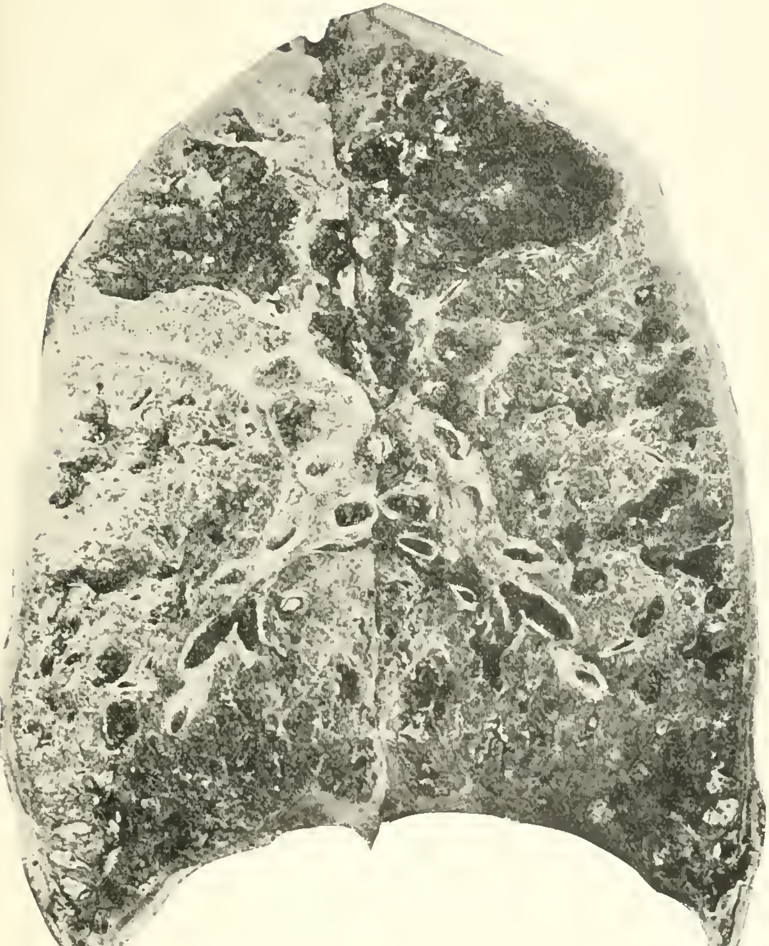

Fig. 10.—Illustrating caseous tuberculosis. Large cavities at the apex and many small cavities throughout the lung. (Tendeloo.)

specific toxemia and the malnutrition which accompany the disease.

Degeneration.—There are three degenerative changes which are commonly found in the bodies of those who die from chronic ulcerative tuberculosis, amyloid, fatty, and cloudy swelling. These three forms of degeneration involve different tissues, but in their selection leave few if any tissues unaffected.

These degenerations are usually explained as being due to the effect of toxins and malnutrition. Just how these factors act is not perfectly clear. Our knowledge of the degenerations of the structures which are affected by reflex stimulation as illustrated in the regional degeneration of the skin, subcutaneous tissue and muscles, offers a basis for thinking that probably the same degenerative tendency might be found wherever tissues stand in a relationship to be affected reflexly by impulses coursing from the lung. If so, then we are led to the conclusion that all organs or tissues which stand in such a relationship with the pulmonary branches of the vagus and the sympathetic divisions of the vegetative system might be so affected. Further, realizing that toxins stimulate the sympathetics centrally, we have a double stimulation of this division which tends to produce the same effect. In this connection, the studies of Crile[4] show that continuous stimulation of nerves by such stimuli as depressive emotional states, pain and toxemias, produce changes in the higher centers. It seems that the way is now open so that we may go a step further than to say that toxemias and malnutrition are the causes of degenerations; and suggest that the cause which operates to produce them, acts, at least partially, through the vegetative nervous system. To this we must also add the possibility of degeneration resulting from changes coming from the chemical stimuli which are produced by altered internal secretions. The work of Fisher in showing that the equilibrium which maintains tissue stability is easily changed, suggests this as an important factor.

Amyloid Degeneration.—Amyloid degeneration, or waxy degeneration as it is called, affects connective tissue and blood vessels. By a gradual change the small arteries and capillaries are involved; their walls are thickened and later become obliterated, thus destroying the function of the part. The effect depends upon the size of the vessel or the number of vessels involved. Amyloid degeneration shows in the liver, spleen, kidneys, and intestinal tract. The affected organs are larger than normal.

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[4] The Origin and Nature of Emotions, W. B. Saunders Company, Philadelphia, 1915.

Fatty Degeneration.—Fatty degeneration has a special predilection for epithelial cells. Whether this is a true degeneration, or simply a deposit of fat, is not yet decided. This degeneration often shows to a high degree in the liver, kidney and heart of those suffering from tuberculosis. Fatty degeneration may be extensive and not interfere greatly with function. In connection with the solution of the cause of this degeneration, I desire to call attention to Fisher's recent studies on the chemistry of tissues.

Cloudy Swelling.—Cloudy swelling of glandular epithelium is common in advanced tuberculosis.

General Congestion.—A varying degree of congestion is present in many of the organs in advanced tuberculosis. This shows in a cyanosis of the skin and as a general congestion of many other organs, particularly those in the abdominal cavity. This congestion may be the result of an extensive active process in the lung, a contraction affecting a large area of lung tissue, or marked pleural adhesions. Conditions which seriously affect the action of the diaphragm result in a deficient expansion of the chest, thus failing to furnish the normal suction action which draws the blood to the heart. This results in a damming back of the blood in the veins. This shows especially in the splanchnics which are markedly congested. As a result of this, we have many of the disturbances of function which are noted clinically in advanced tuberculosis.[5]

Changes in Nerves.—Changes in nerves are also noted in tuberculosis. Aside from the general effect of toxins upon the nervous system as a whole, changes take place in the particular nerves which take their origin from the segments of the cord which receive the impulses from the diseased pulmonary tissue. This is evident clinically, although it has not been reported on in detail pathologically. This irritation clinically at times amounts to a marked neuritis.[6] Since first noting this, nine cases of brachial neuritis all on the side of the lung with the severest process, have come under my observation. See page 163.

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[5] Pottenger: Enteroptosis and Altered Function of the Diaphragm Resulting from Intrathoracic Inflammations, New York Medical Journal, December 16, 1911.

Changes in Muscles.—Changes in the muscles[7] are found not only as a result of the general malnutrition and toxemia, but as a result of a regional reflex. The reflex degenerative changes found in these muscles were described as follows: "One finds in such muscles both degenerative and regenerative changes. Sometimes the stripes are indistinct and, at times, they are entirely wanting. The fibers take the stain poorly and in some instances are destroyed. In many preparations one sees an increase in the nuclei of the cells and slight signs of regeneration."

This change, as noted clinically, is an increased tension when the disease in the chest is acute and a flabbiness of the muscles when the inflammation has once passed over into the chronic state.

This follows the same law as just mentioned in describing the changes in the nerves and is first a response to the stimulation from the irritated nerve and secondarily a degeneration resulting from overwork or the disturbance of the trophic impulses.

Changes in the Skin and Subcutaneous Tissue.—Atrophy of the skin and subcutaneous tissue takes place as a result of reflex segmental irritation, the same as occurs in the muscles. This is regional in character and is entirely separate from the general degeneration which occurs in chronic tuberculosis.

Changes in Blood Vessels.—Changes in blood vessels are also common and of vast importance from the clinical standpoint. As mentioned above, amyloid degeneration affects the vessels of the various organs, such as the kidney, liver, spleen, pancreas and intestines and interferes with their proper function. As a result of toxic irritation, the same as is noted after typhoid (Thayer), a thickening takes place in the arteries,[8] as noted on palpation of the radials. This shows itself especially in chronic tuberculosis when the disease has existed for a long time and been pouring out toxins in small quantities over a period of months or years. The arteries are often smaller than normal, as has been mentioned by Martius[9] and Beneke,[10] as a predisposing factor in infection, but which I have explained as being a result of the diminished inspiratory act resulting from the disease. See page 301.

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[6] Pottenger: Chest and Shoulder Pains in Tuberculosis, Transactions of the American Climatological Association, 1912.

[7] Pottenger: A New Physical Sign Found in the Presence of Inflammation of the Lungs and Pleura, Journal of the American Medical Association, March 6, 1909; and "Die Rigidität der Muskeln und die leichte Tastpalpation als wichtige Zeichen zur Erkennung der Lungenkrankheiten," Deutsche Medizinische Wochenschrift, Nr. 16, 1910.

[8] Pottenger: The Effect of Tuberculosis on the Heart, Archives of Internal Medicine, October, 1909, vol. iv, p. 306.

Tubercle bacilli often invade the vessel walls both from within and without, and not uncommonly tubercles form within the walls and rupture, causing hemorrhage. At other times this rupture allows bacilli to escape into the lumen and scatter infection by way of the blood stream. If many bacilli escape at one time, generalized miliary tuberculosis results; if a few, solitary or multiple tubercles form.

Non-pulmonary Tuberculosis.—Many organs and tissues outside of the lungs are at times affected with tuberculosis, the process being one which either complicates a pulmonary infection or one entirely independent of it. The pathology of these various lesions is the same except as the process adapts itself to the peculiar characteristics of the different kinds of tissue in which it is found.

Nasal, Lingual, Tonsillar, and Pharyngeal Tuberculosis.—Tuberculous processes are occasionally found in the nasal mucous membranes, tonsils, pharynx and tongue, and quite often in the larynx. These may be either proliferative, degenerative or mixed in character. Lesions of the nasal mucous membranes, pharynx and tonsil in the writer's experience have been for the most part of the degenerative type and have shown little tendency to heal. Lesions of the tongue have been for the most part of the proliferative type with marked tendency to heal; while lesions of the larynx have been of the degenerative, proliferative and mixed, and, as a rule, have shown a tendency to heal in nearly all instances where the disease in the lung was taking a favorable course. Figs. 11*A* and 11*B* illustrate two tuberculous ulcers on the tongue of the same individual which healed after two and one-half years' treatment. Fig. 11*C* shows an ulcer on the dorsum

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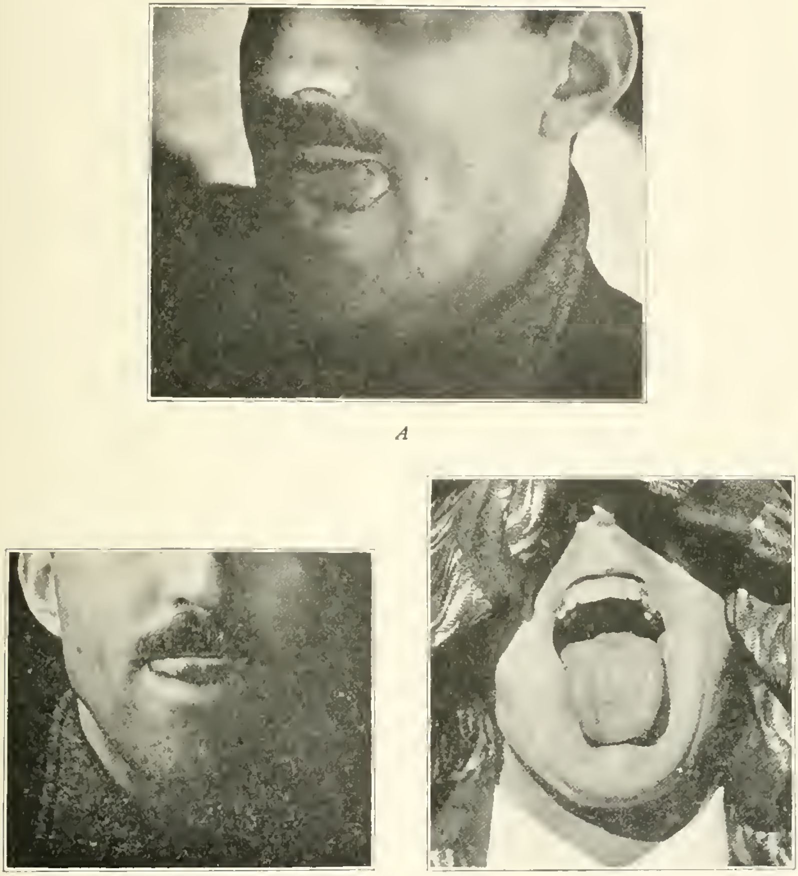
[9] Pathogenese innere Krankheiten.

[10] Quoted by Sokolowski, Klinik der Brustkrankheiten, Berlin, 1906, vol. ii, p. 237.



A

B

C

Fig. 11.—Illustrating tuberculous lesions of the tongue, *A* and *B* in the same individual, healing after two and one-half years' treatment; *C*, on the dorsum of the tongue of an individual suffering from active advanced tuberculosis; no signs of healing shown, patient dying of the disease a few months later.

of the tongue which failed to heal, the patient dying of advanced tuberculosis.

I have never seen a primary lesion affecting any of these tissues, although such is sometimes reported in literature, and is possible.

Laryngeal Tuberculosis.—Any portion of the larynx may be the seat of tuberculous infiltration and ulceration. The focus is usually subepithelial in character but may be deeper in the tissues. The lesion may be slight or it may be accompanied by an extensive destructive process. It may show either as an infiltration or ulceration. The most serious processes are perichondritis and chondritis, which often lead to softening and purulent degeneration.

The interarytenoid space, the arytenoids, and the cords are the most common seats of infection. This represents two different types of tissue, the arytenoid region being extremely well supplied with lymphatics while the cords are not. In 61 cases reported by the writer,[11] the arytenoid region and the cords were affected 45 times or in 73.77 per cent. Besold's cases[12] showed 316, or 63 per cent infections of the arytenoid region and cords in 498 cases.

Tuberculosis of the larynx is nearly always secondary. Cases are now and then diagnosed as primary because of a failure to discover the original focus. The fact that the disease is practically always found as a complication of pulmonary tuberculosis suggests that this organ is not a natural site for the disease as it spreads through the body by means of the blood. It also suggests that the infecting bacilli come from the lesion in the lung, whether by way of the lymphatics, or by direct infection from sputum is not determined. Richard Lake[13] takes the view that the mode of entry is nearly always by surface infection and states that there is no direct lymphatic connection to which the infection can be traced. It would seem that this view is rational; and it harmonizes with the idea of the secondary infection of the intestines being of surface origin.

Tuberculosis of the larynx is common. Postmortems reveal laryngeal infection in about 50 per cent of those dying of tuberculosis. Clinical observations show about 30 per cent of frank involvement, while painstaking examination raises this percentage to forty or forty-five. Besold and Gideonson report 498 or 26.73 per cent in a material of 2,018 cases. The writer reported 61 cases or 29.3 per cent of well marked clinical tuberculosis of the larynx in a material of 208 patients.

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[11] Prognosis and Treatment of Tuberculous Laryngitis: An Analysis of Sixty-one Cases, California State Journal of Medicine, October, 1908.

[12] Besold and Gideonson: Pathologie und Therapie der Kehlkopftuberkulose, Berlin, 1907.

[13] Laryngeal Tuberculosis, William Wood & Company, New York, 1905.

Tuberculous foci in the larynx are accompanied by collateral inflammations of variable severity. Sometimes this amounts to only a hyperemia; at other times there is a marked edema; and, still again it shows as a purulent exudate with destruction of tissue.

Laryngeal lesions of the proliferative type show a marked tendency to heal when properly treated, especially in cases where the pulmonary disease is not too active. Ulceration, on the other hand, while often healing, shows a tendency to progress.

Tuberculous Pleurisy.—Tuberculous inflammation of the pleura exists both as tuberculous foci and as collateral inflammation. Tuberculous pleurisy is usually spoken of as being of two kinds, the exudative and the dry, although this is not strictly correct from the pathological standpoint, for even the dry is due to a fibrinous exudate. The pleura may be the seat of miliary tuberculosis.

Tuberculous pleurisy then, is an exudative inflammation which may be serous, sero-fibrinous, fibrinous, hemorrhagic, or purulent, in character. The exudate is poured out into the pleural sac either as a result of a tuberculous process affecting the pleura itself or the subpleural tissue of the lung. It may consist of a few drops of fluid or of an exudation sufficiently large to fill the pleural space. It may affect one side or both. If of the fibrinous character, it may result in the formation of a few tender fibrils of tissue or the development of connective tissue covering the entire lung surface even to the thickness of an inch.

Purulent exudation infected by pus organisms is rare in tuberculosis, even in those cases of spontaneous pneumothorax where a necrotic tubercle breaks through into the pleural cavity. This fact goes to substantiate the theory that necrotic processes in tuberculosis are not as a rule infected by or due to pus organisms. When infection occurs it is probable that an old infected cavity, rather than a recent necrotic process breaks into the pleura. We may have a purulent exudate that is not infected, due to cheesy subplural masses breaking into the pleural cavity. This is considered as always being of tuberculous origin.

Absorption of either serous, sero-fibrinous, or fibrinous exudate may take place. The subpleural lymphatics have great power of absorption; but if the amount of exudation is large it compresses the lymphatics. If it is fibrinous and covers much of the pleural surface it closes up the stomata and so interferes with absorption. Absorption of fluid is often hastened by the withdrawal of a comparatively small amount of fluid. It is probable that this acts by relieving the compression of the lymphatics, thus permitting them to resume their function. Fibrinous pleurisy can also be absorbed, although it usually leads to adhesions between the visceral and thoracic pleuræ.

Often following pleurisy a large amount of fibrous tissue is formed, not only on the pleural surface but extending into the lung substance, which contracts and causes much deformity of the lung. When the process involves the diaphragmatic pleura, the action of this important muscle of respiration is interfered with causing a chain of clinical symptoms (see Vol. II, Chap. XXV). Often the pericardium partakes of this inflammation resulting in an embarrassment of the heart. The motility of the affected lung is decreased, a compensatory emphysema results, and a degree of strain is thrown back upon the heart.

Tuberculous Pericarditis.—Tuberculous inflammation of the pericardium may result from tubercles in the pericardium itself or from a collateral inflammation, resulting from a tuberculous process near the pericardium, when there is no specific process in the sac itself. It may be an extension from the pleura.

The process may show the same characteristics as pleurisy, the exudate being serous, sero-fibrinous, cellular, or purulent. Purulent pericarditis is extremely rare.

At times the pericardium takes upon itself very severe changes. It may become thickened, the cardiac portion sometimes appears spongy. The two surfaces occasionally become adherent and obliterate the entire sac. This condition is followed by myocarditis and atrophy of the heart muscle.

When the exudate is serous in character it may become absorbed of its own account the same as pleuritis.

Tuberculous Peritonitis.—Tuberculous peritonitis shows all the characteristics of tuberculosis of serous membranes.

Miliary tuberculosis is a very common variety and at times is found unexpectedly when operating in the abdominal cavity. At times the entire peritoneum is studied with tubercles, all apparently of the same age, showing that they are of common origin. Their distribution and characteristics are such that they can only be of hematogenous origin. This form shows little or no tendency to necrosis and is usually not accompanied by exudation unless one of a slightly fibrinous character. Hyperemia usually surrounds the tubercles. Being of hematogenous origin and of low virulence, there is almost no collateral inflammation. This form corresponds to the disseminated fibroid tuberculosis of the lung both in origin and virulence.

Another form is that accompanied by serous exudate. This has been looked upon as a favorable form, especially after operation, since Königs' report in 1884. It is also favorable without operation, the same as serous pleuritis. Many reasons have been assigned for the cure of peritoneal tuberculosis which takes place following operation. Some have explained it as being due to the light entering the abdomen, but such does not seem to be rational. Others have thought the handling of the intestines causes a hyperemia which favors healing. It seems that the change in intra-abdominal pressure is also probably a factor. Anything causing increased hyperemia and increased lymph flow is valuable. But in finding a reason for cure following operation we must not forget that cure follows without operation in probably as large a percentage of cases as it does after operation.

This form of tuberculosis shows nodules of various sizes, some of which are often necrotic. There may be considerable formation of new tissue with adhesions and a matting together of the intestines. All of the abdominal organs are subject to disturbed function and sometimes to tuberculous infection as a result of the peritoneal changes or as a part of the same infection. The peritoneum is subject to all the different forms of exudation that other serous membranes are, serous, fibrinous, sero-fibrinous, cellular, and purulent.

When purulent peritonitis is present, the fluid may rupture into the intestines or break through the abdominal wall. When this happens it usually occurs in the region of the umbilicus. This occurs most commonly in children.

Not only serous but fibrinous exudate may disappear. Even adhesions may be absorbed, leaving no trace. This is analogous to what happens in the absorption of adhesions after ordinary operations in the abdomen.

Tuberculous Enteritis.—Tuberculosis of the intestinal tract is a very common complication of advanced pulmonary tuberculosis. The process may cause distressing symptoms or it may be present without being suspected during life. The most frequent seat of tuberculous infection is in the lower ileum, cecum, appendix, and ascending colon, although any portion of the gut may be involved. Even the stomach shows tubercles occasionally. It is interesting to note that the most common seats of infection are the areas favored by stasis of the contents of the bowels, thus favoring the theory of contact infection. The mucous membrane of the lower portion of the large bowel is at times the seat of ulceration, at other times it is involved in a fistulous process from a tuberculous abscess in the deep tissues around the rectum. The process which leads to the formation of a pulmonary cavity and that which leads to the formation of a rectal fistula is the same except that it affects different tissues.

The tuberculous process in the intestines may belong either to the proliferative or degenerative type, or may be a mixture of the two. Ulceration is a common form of the infection and is, occasionally, the cause of perforation. At times, however, there is a tendency to the generation of new tissue and we find the walls of the intestines enormously thickened. At times the gut appears like a thick-walled tube. Again, there is a local thickening with constriction at intervals along the course of the gut; the portions between, becoming thin and greatly dilated because of the distention that takes place as a result of the bowel contents and gas collecting between the obstructions. The thickenings of the gut sometimes cause stricture of the lumen.

It was formerly thought that bacilli in the feces were diagnostic of tuberculous enteritis, but we now know that a proliferative process might be present without discharging bacilli in the bowel. Three hundred examinations of stools made in the laboratory of the Pottenger Sanatorium showed tubercle bacilli present in the stools in about one-half of the cases of advanced tuberculosis, when the greatest care was taken to avoid the swallowing of sputum. Preparatory to the test, the patient was especially instructed not to swallow any sputum. Then a dose of castor oil was given. After the effects of the oil had passed, the stool was saved for examination. The stool was homogenized by shaking in the shaking machine and a smear was taken for examination. (See Chapter XX.)

Tuberculous enteritis of the ulcerative type shows very little tendency to heal although we do sometimes find evidence of its having occurred. These ulcers sometimes perforate allowing the bowel contents to escape into the peritoneum.

Diarrhea is not a necessary accompaniment of ulceration of the bowel. This depends much on the location of the lesion. Some of the most extensive ulcerations in the region of the cecum that I have seen, postmortem, were accompanied by a normal stool, others by a soft stool, and still others by constipation. When the lower end of the large bowel is the seat of ulceration severe diarrhea is apt to be present.

The question of primary infection of the bowel has received careful attention since Koch's[14] announcement of the view that human and bovine bacilli differ. Out of the great mass of experimental study the conclusion has finally been drawn that bovine bacilli are a menace to human beings and accountable for a considerable amount of tuberculosis among children, particularly the non-pulmonary forms. (See Chapter II for further discussion of this point.) In order to produce infection through the alimentary canal it is not necessary that the bacilli should produce a primary lesion in the intestinal wall. The bacilli when first taken in, pass through the walls of the gut and are carried by the lymph to the glands of the mesentery and even to the mediastinum; or may be carried by the lacteals to the thoracic duct and emptied directly into the blood stream to be strained out in any organ.

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[14] Report of the International Congress on Tuberculosis, London, 1901.

The difference in the action of the intestinal wall toward bacilli taken in in early life and those swallowed with tuberculous sputum requires some consideration although I have never seen this question discussed. Why do the bacilli pass through the intestinal wall of children, or we might say, of the non-tuberculous in general and leave no trace, while they produce infiltrations and ulceration in the walls of the intestines of those suffering from tuberculosis? Is the difference in behavior due partly or wholly to the great numbers of bacilli swallowed by the sufferer from advanced pulmonary tuberculosis in comparison with the few which enter the intestinal tract of the non-tuberculous? Is it not probable that the difference is due more to the sensitization of the cells in the intestinal wall in the one as compared with the lack of it in the other? Is it not probable that it is the same experiment as that of Koch which showed the difference between the inoculation of the tuberculous and the healthy guinea pig? Prior to the time that infection has occurred and the cells have been endowed with specific defensive powers (sensitized) the bacilli pass through the mucous membranes with little opposition and are carried to the regional lymph glands; but, after infection has taken place, the bacilli which enter the tissues of the gut set up a reaction with the local cells which results in an inflammation; an infiltration resulting if the bacilli are of low virulence, and an ulceration if they are of a more virulent strain. This cell sensitization is probably a factor in all metastases and superinfections, such as those in the lung and larynx. This subject will need much careful study for it will probably explain many of the vagaries noted in extensions of the tuberculous process. (See Chapter III for a more complete discussion.)

Tuberculosis of the Liver.—The liver may be the seat of miliary tubercles, or it may contain cheesy degenerated foci, or may show a general fibrosis (tuberculous cirrhosis). It may be infected through the alimentary canal or through the blood from a focus in the lung or other parts of the body. Some observers report finding tubercles in nearly 90 per cent of livers of those dying of chronic tuberculosis.

Aside from the specific tuberculous infection, the liver is the seat of non-tuberculous degeneration in chronic tuberculosis. Its cells show cloudy swelling of various degrees and fatty degeneration is extremely common. When fatty degeneration is present miliary tuberculosis may be present and be overlooked.

A perihepatitis is often present especially involving the diaphragmatic portion of the liver. This results as an extension of an inflammation from the diaphragmatic pleura.

Tuberculosis of the Spleen.—Tuberculosis of the spleen is sometimes of hematogenous origin and takes the form of miliary tuberculosis; again, it may be of lymphogenous origin in which case the foci are apt to be of variable size and may be necrotic. When of lymphogenous origin the bacilli are implanted in the perivascular lymph spaces, while the hematogenous type is found in the pulp of the organ.

Tuberculosis of the Glands.—We recognize the spreading of tuberculosis through the lymph channels in connection with the pathology of all the organs of the body. We see, too, that it is a form of spreading which favors a virulent metastasis because the bacilli escape from the tubercle into adjacent tissue which has been injured by toxins in which the lymph movement is slow; and the bacilli may be thrown into the lymph spaces in numbers and find themselves surrounded by conditions favorable for growth. An exception to this is found near the hilus where bacilli gain entrance to the large lymphatic trunks and produce infection with difficulty; the type, as a rule, being abortive in character. The condition as a rule, however, is the opposite of that which obtains when bacilli enter the blood stream. Here the rapidly flowing blood scatters the bacilli to different parts of the body and prevents their heaping up in any one focus. They are injured by the antibacillary elements of the blood and become implanted in healthy tissue which possesses specific cellular defense. The result is that tuberculosis, spread by the lymphatics, is an implantation of numbers of bacilli in each focus, followed by rapid multiplication and necrosis, while that spread by the way of the blood, unless enormous numbers of bacilli gain entrance to the blood stream, is an implantation of isolated or few bacilli in the capillaries of an area of greater or lesser extent, followed by a slow multiplication which favors stimulation of the cells to the formation of new tissue. Tuberculosis which spreads by the lymphatics then, is, for the most part, accompanied by necrosis, more or less toxemia, considerable collateral inflammation, and has a tendency to rapid destruction of tissue; while that which spreads by the blood stream is comparatively mild, non-virulent, accompanied by slight toxemia and shows a tendency to produce new tissue and either becomes encapsulated or runs a chronic course. The exceptions to this are acute miliary tuberculosis of hematogenous origin where the inoculation is so massive that it destroys the patient; and that non-virulent infection of the lungs which takes place through the large lymph trunks near the hilus.

Aside from the part that the lymphatics play in the dissemination of bacilli through the body, the walls of the lymphatics themselves, and particularly the glands, may be infected by tuberculosis. Occasionally, a miliary tuberculosis results from a rupture of a tubercle in the walls of the thoracic duct.

Tuberculosis is primarily a lymphatic disease. No matter in what manner bacilli gain entrance to the body, they find their way to the lymphatics and usually to the neighboring lymph glands, there to set up the disease. (See page 84.) It is from this focus of infection that the disease spreads to other tissues either adjacent or distant. While tuberculosis of the lymph glands is, in itself, not so serious; yet when we consider that it is the cause of the metastases in other parts of the body, it assumes great importance.

Tuberculosis of the lymph glands is a very different pathological lesion from tuberculosis which spreads in the tissues adjacent to other tubercles by way of the lymph spaces. The former is usually a non-virulent infection, the latter a virulent one. The difference is that the former focus is one constantly bathed with fresh lymph, consequently the bacilli are kept of low virulence, while the focus which spreads by a heaping up of bacilli in lymph spaces is surrounded by stagnated lymph and the bacilli are left to multiply rapidly, producing a virulent process.

The glands commonly infected are the cervical which drain the nasopharynx, gums and tonsils; the mesenteric; and, most commonly, the peribronchial. Figs. 13 and 14, pages 93 and 94, show the relationship of the structures in the pharynx and oropharynx to the deep cervical lymphatic glands.

The glands are sometimes enormously enlarged. They may present any degree of inflammation, sometimes being almost free from accompanying symptoms, and, again, showing signs of acute inflammation. Sometimes the center becomes necrotic, and breaks down, discharging its contents either externally or into the adjoining tissues. Rupture into a vein or the thoracic duct may cause general miliary tuberculosis.

After the glands have become necrotic, lime salts may be deposited forming, in the lung, the "lung stones" which patients sometimes expel.

Inflamed glands may not go on to caseation, but become fibroid in character. Such glands usually lose their functionating power the same as the necrotic and calcareous ones.

CHAPTER II.

THE SOURCE AND ROUTES OF INFECTION AND THE PRIMARY FOCUS.

In discussing the theme of infection we enter upon the consideration of questions which have received the attention of many able investigators but still remain in doubt.

Source of Infection.—Until Koch made his announcement at the British Congress on Tuberculosis in 1901, the identity of the bovine and human bacillus had been seriously questioned among men of scientific reputation only by Theobald Smith; and their intertransmissibility was generally accepted. Not only was the intertransmissibility of these two types of bacilli considered as a fact, but they were looked upon as being of almost equal etiological importance in the production of clinical tuberculosis. After this announcement, however, many workers in all lands spent years in earnest investigation of this subject, finally arriving at certain conclusions which seem to satisfy the scientific world at large. Koch's theory, as he first announced it, confirming that of Theobald Smith, that there is a difference between bovine and human tubercle bacilli, is now generally accepted; but Koch's further statement that bovine bacilli are of little or no consequence in the causation of human tuberculosis, has not been substantiated. The conclusion has been reached, however, that the human bacillus is the main source of human tuberculosis and that the bovine bacillus is accountable for a much smaller proportion of the disease than we formerly believed. While it is difficult to say how much tuberculosis is of bovine origin and how much of human origin, yet pathologists who have studied the question carefully now agree that about eleven-twelfths of all tuberculosis in man is caused by human bacilli and about one-twelfth by bovine bacilli; and that bovine infection, as it occurs, predominates in childhood, while the human infection, although accountable for a large per cent of disease during childhood is accountable for nearly all disease of adult life.

Park and Krumwiede[1] analyzed 1,038 cases of tuberculosis, which had been carefully studied, with the following results:

AGE PERIOD	PER CENT OF HUMAN TUBERCULOSIS	PER CENT OF BOVINE TUBERCULOSIS
0— 5 years.............	73.5	26.5
6—11 years.............	75.	25.
16 years and over.........	98.69	1.31

In this connection the following table from the Imperial German Board of Health is also interesting. It presents an analysis of 1,400 investigated cases.

	TOTAL NUMBER INVESTIGATED CASES	TYPES		PER CENT OF ALL CASES DUE TO BOVINE TYPE	
		HUMAN	BOVINE	IN ADULTS	IN CHILDREN
Tuberculosis of the lungs...	811	807	5	0.66	0.
Tuberculosis of the bones and joints..............	99	95	5	6.66	4.3
Meningeal tuberculosis....	33	30	3	0.	10.34
Generalized tuberculosis...	178	147	33	2.5	23.18
Tuberculosis of the cervical glands..................	167	120	47	5.8	40.7
Tuberculosis of the mesenteric glands............	112	78	35	12.0	51.0
Totals................	1400	1277	128		

The studies of Eastwood and Griffith[2] are very interesting in that they have made a special study of the type of infection in bone and joint tuberculosis. Basing their classification upon cultural characteristics and the virulence of the bacilli as shown upon rabbits, they examined a total of 261 cases and found the human type of bacilli in 196, bovine in 55, and a bacillus which they were unable to classify, in 10.

I desire to emphasize in this connection the fact that the bovine type of bacillus disappears very rapidly from clinical tuberculosis after the tenth year, indicating, as I have expressed elsewhere, that the bovine bacillus produces infection only during early child life; or, that it probably changes its characteristics with growth upon human soil for many years, and assumes the characteristics of the human bacillus.[3]

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[1] The Relative Importance of the Bovine and Human Types of Tubercle Bacilli in the Different Forms of Tuberculosis, Journal of Medical Research, December, 1911.

[2] The Characteristics of Tubercle Bacilli in Human Bone and Joint Tuberculosis, Journal of Hygiene, vol. xv, No. 2, 1916.

The results of the examinations reported by Eastwood and Griffith are shown in the following tables:

TYPES OF TUBERCLE BACILLUS AT DIFFERENT AGE PERIODS

AGE PERIOD	NUMBER OF CASES	HUMAN	BOVINE	ATYPICAL
0—5 years...........	47	31	14	2
5—10 years...........	108	75	31	2
10—16 years...........	62	52	7	3
16—25 years...........	15	12	3	..
Over 25 years..........	29	26	..	3
Total................	261	196	55	10

The percentage of "bovine" cases are:

All ages	(55 out of 261)............	21.1 per cent
Under 10 years	(45 out of 155)............	29.0 per cent
Over 10 years	(10 out of 106)............	9.4 per cent

We must recognize the fact that our methods of determining the difference between bovine and human infection are not absolutely reliable; yet we cannot help noting that the results obtained by different observers agree fairly well. It is also suggestive that all find little bovine infection in adult life.

These same writers have made a study of the types of bacilli occurring in the genitourinary tract and I will quote their summary.

"Seventeen cases were examined, the disease affecting the genital organs in nine instances (seven testicles, one salpinx, one prostate) and the urinary tract in eight.

"The bacilli obtained were of 'human' type in fourteen cases and 'bovine' in three.

"The three 'bovine' cases were affections of the kidney in persons aged, respectively, 25, 19 and 20 years."

Griffith[4] further reports an analysis of results obtained from the investigation of sputum of 212 patients suffering from pulmonary tuberculosis, with the following results:

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[3] Pottenger: The Relation of Bovine Infection in the Child to Clinical Tuberculosis in the Adult, Southern Medical Journal, November, 1915.

[4] Investigations of Strains of Tubercle Bacilli Derived from Sputum, The Lancet, London, No. 4,831, vol. cxc, April 1, 1916.

	Number of Cases	Type of Tubercle Bacilli Isolated		
		standard human	atypical human	standard bovine
England	139	135	2	2
Scotland	73	70	2	1
Total	212	205	4	3

In discussing this question Griffith says:

"In this country, therefore, pulmonary tuberculosis which has arrived at the ulcerative stage is but very rarely referable to tubercle bacilli of bovine type. And elsewhere than in Britain an even smaller proportion of the sputa of persons suffering from pulmonary tuberculosis has yielded bacilli of bovine type. Out of 736 cases tested by foreign investigators 3 only yielded bovine tubercle bacilli; and further, in 2 of these 3 the bovine bacillus was only sparsely mixed with preponderating bacilli of human type, while in the third case proof was not afforded (only a single examination of the sputum having been made) that in this instance also both types of tubercle bacillus were not coexistent in the lungs of the patient."

While such apportionment of the bovine and human infection in man seems to be fairly well accepted, yet it comes in conflict with other pathological ideas which seem to be fairly established. We are taught today that clinical tuberculosis in adult life is largely an extension from an infection which takes place in early child life (Römer). If this is true, and our knowledge of the disease supports the theory, what is there to hinder this metastatic infection in later life from being of either bovine or human origin; and, according to the data quoted above, why is not adult tuberculosis more largely of bovine type, unless mutation of type takes place? Children unquestionably take in both types of bacilli and are infected by the same; and, as yet we do not know that the resulting infections differ to any great extent, or even at all, in their subsequent pathological changes or clinical course.

Bovine Infection Cannot be Differentiated From Human Infection Either by Localization or Character of the Lesion.—Many observers, among whom is the writer, have been inclined to believe that those forms of tuberculosis which seem to predominate in organs other than the lungs, and which are particularly common in early childhood, are most probably due to bovine infection. We were inclined to believe that the typical human infection is tuberculosis of the lungs; and to look upon other forms of tuberculosis, such as those involving the bones, joints, glands, meninges, intestinal tract and peritoneum as being atypical; and, since these forms are more common in childhood than in adult life, to consider them as belonging to the bovine type of the disease. Believing that the bovine bacillus is less virulent for man than the human type, we reasoned that it would probably produce an atypical disease. Further study, however, seems to make this untenable.

As near as we know at the present time, tubercle bacilli, whether of human or bovine type, no matter how they are taken into the body, whether it be through the upper or lower respiratory tract, through the tonsils, pharyngeal mucous membranes, or intestinal tract, find their way into the lymph spaces, and, for the most part, into the lymphatic glands and establish a primary lymphatic tuberculosis. As exceptions to this, are such cases of tuberculosis as are produced by the direct inhalation of bacilli into the finer air passages (if such occurs), setting up a local infection in the pulmonary tissues at the point of inoculation; and, infections produced by bacilli passing through the intestinal mucous membranes and being taken up by the lacteals and poured into the thoracic duct from which they are emptied into the blood stream and carried directly to the capillaries of the lung, or other organs where they find lodgment and produce infection. The bacilli which would take either of these latter courses might be either of the bovine or human type. They could be taken in either by inhalation or ingestion, and their implantation could occur wherever the blood stream is sufficiently retarded for the bacilli to find time to enter the tissues. It is in this manner that we recognize the possibility of primary infection of organs other than the lymphatic glands taking place.

In the primary lymphatic tuberculosis, as far as we know, there is no clinical method of distinguishing tuberculosis of the bovine type from tuberculosis of the human type. This is left to laboratory investigation and animal experimentation. We know that both bovine and human bacilli can affect these glands alike, and that up to this point the paths of infection are undoubtedly the same. From this point the bacilli are carried by the lymph stream; by wandering cells; or, breaking into a blood vessel, are transported to other parts of the body, either adjacent or remote. Such a course is as likely to be followed by bovine bacilli as by human bacilli. If bacilli gain access to a vessel which leads toward the heart the metastasis from the lymph gland would most probably be in the lung because the bacilli would most likely be strained out by the capillaries in the lung. If, on the other hand, the bacilli should not be strained out in the lung but pass on through the left heart; or, if they should pass directly into the systemic circulation (as by passing into the pulmonary veins), they might be strained out in any part of the body, the intestines, liver, kidney, bones, joints, meninges, or any other organ. Where this implantation will occur depends upon where the blood current is sufficiently retarded to permit the bacilli to become implanted. This offers a basis for primary infection of many organs.

There has been considerable confusion of opinion as regards metastatic tuberculosis. While it is probable that the disease may spread from the primary focus by way of either the lymph stream, wandering cells, or the blood stream, it is equally probable that most metastases which are responsible for a new area of infection must take place through the blood stream. In the case of metastases distant from the primary focus in such organs as the kidney, joints, bones and meninges, this is evident. In the lung, some writers think the lymph stream and wandering cells are the important factors, while others think the bronchi are most important.

Important as bearing upon this is the theory which is accepted by such students of the subject as Tendeloo, that bacilli may be transported against the lymph stream. While the general course of the lymph stream in the lung is toward the hilus glands, with the alternate contraction and expansion of the pulmonary tissue, it is quite possible for the lymph to be forced backwards in its course and carry bacilli with it.

It is not improbable that those infections of pulmonary tissue which take place around and extend out into the lung from the hilus, might be implantations which have taken place through the lymph stream. At this point the lymphatic trunks are larger, the current retarded, but implantation is not favored. This is the tuberculosis which is detected by the x-ray when the apex is not involved. It is quite different, however, from the usual clinical tuberculosis which begins at the apex, whose implantation has been not in contiguous tissue, but in tissue widely separated from the primary source of infection. In this latter infection the blood stream alone solves the problems connected with the distant implantation.

While we have been inclined to look upon the non-pulmonary forms of tuberculosis as being different from tuberculosis of the lungs,—as being a milder infection,—and to suppose that bovine bacilli, not being so well adapted to human soil, are more apt to produce a milder infection, our newer knowledge brings this theory into question. The reason we look upon tuberculosis of the lungs as being a more virulent process than tuberculosis in these other organs is partly, at least, because we are comparing different conditions. The infection which takes place through the blood in nearly all instances, whether it be a primary or a metastatic infection, is at first benign; exceptions being in those instances where a large number of bacilli are poured into the blood stream at once and produce a widespread miliary tuberculosis. This, too, would be benign were it not for the great numbers of bacilli, causing widespread infection. Only the mass of the infection makes it serious.

When tubercle bacilli break into the blood stream in small numbers they are so diluted and so acted upon by the protective substances found in the circulating blood and so opposed by the defensive properties of the tissue cells in which they attempt to settle that, for the most part, they are destroyed. In those instances where they succeed in producing an infection, this infection is caused by such bacilli, probably often clumps, as have been able to escape the antibacillary action of the blood elements; but which, we are justified in assuming, have been reduced in virulence;

so that we have an infection caused by a few bacilli; and those bacilli, of moderate virulence. Most of these infections being metastases, the implantation takes place in tissues whose cells are sensitized and able to put up a specific defense. The result is that, instead of a rapid multiplication of bacilli with necrosis and caseation, and a rapidly developing activity, we have an infection produced by an organism which is reduced in virulence and barely able to exist, consequently we have an irritation with resultant new tissue formation, and a focus fibroid in character. Thus we can see that nearly all early infections are of this type. Infection in such organs as the bones, joints, kidneys, meninges and visible glands, is usually discovered soon after infection starts, at a time when the focus is small. Tuberculosis in the lung, however, is not, as a rule, discovered during this early stage. The small focus produces either no recognizable symptoms or so few symptoms that its presence is not suspected; and there it remains, for years often, before it becomes a clinical entity.

As soon as the bacilli begin to multiply, however, toxins escape into the adjacent tissues, a collateral inflammation ensues and the whole process assumes a state of active inflammation. Symptoms now appear and the disease is recognizable. If it goes on to a wide extension into adjacent tissues, as it often does, we have an advanced process and one that is not easy to heal. This is the picture of the process in the lung which is being compared with the small focus in other organs. If the bacilli become active, multiply, and produce necrosis and caseation in other organs, then we have marked symptoms on their part, the same as on the part of the lung.

It does not seem then, from the data at hand, that we are justified in saying that there is a difference in the type of bacilli that produce the disease in such locations as bones, joints, meninges and glands, from that produced in the lung. It is more probable that we are comparing slight activity in a small fibroid lesion in one instance with a rapidly extending or more marked activity in the other; and it further seems that when we add the differences in the character of the tissue and the function of the organs involved that we probably have a satisfactory explanation for the difference in the character of the disease in the different parts. It may be that there are certain points of election, that there is a certain soil that is particularly suitable to the growth of the bovine bacillus and certain tissues and soil that are more suitable to the human bacillus, but of this we cannot be sure. It is also not improbable that long growth on unsuitable soil is followed by adaptive changes on the part of the bacillus.

On the other hand, we are justified in assuming that the bovine bacillus can be taken into the system in the same manner as the human bacillus, that it must pass through the same barriers to get into the tissues, and that it probably takes the same course in forming metastases; consequently, it seems but natural that there should be great difficulty in differentiating the infection as it is produced by the two bacilli by clinical methods. It is also quite possible that bovine bacilli may change their morphology and their characteristics of growth to suit the new soil. *It would not tax our credulity in the least to believe that bacilli of the bovine type might be taken into the tissues during childhood, and by adapting themselves to the human tissues for many years, might assume characteristics of the human bacillus by the time they were ready to extend to the lung or other tissues and produce clinical disease.* In fact this process of adaptation might be the reason for a dormant process in a gland taking upon itself activity, and the bacilli which had previously been held in check in their growth, finally becoming suited to the soil and multiplying, inflaming the tissues and escaping to form metastases in other parts of the body.[5]

Incubation Period in Tuberculosis.—If we knew more of the incubation time in tuberculosis it would help somewhat in our determination of the source of infection. Bartel[6] has described a lymphatic stage of tuberculosis in which the infectious organisms are viable, yet have not produced histological tubercle. Römer and Joseph[7] have found an incubation period of three and a half months in guinea pigs. How long this stage is in man we do not know, yet we are led to infer that it is a long one.

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[5] Pottenger: Some of the Anatomic-Pathologic Problems in Tuberculosis, Transactions of the American Climatological Association, 1915; also, The Relationship of Bovine Infection in the Child to Clinical Tuberculosis in the Adult, Transactions of the Seventh Pan-American Medical Congress, San Francisco, June 17-21, 1915, Southern Medical Journal, November, 1915.

[6] Die Bedeutung der Lymphdrüse als Schutzorgan gegen die Tuberkuloseinfection, Wiener klinische Wochenschrift, Nr. 4, 1905.

[7] Prognose und Inkubationsstadium bei experimentelles Meerschweintuberkulose, Berliner klinische Wochenschrift, 1909, Nr. 28.

The fact that bacilli can pass through the tissues without causing local infection also makes the riddle more difficult to solve. Mucous membranes are readily penetrated and bacilli are carried to lymph glands, at the time of the primary inoculation. This action on the part of the bacillus differs from that which occurs in the formation of metastases after specific cell sensitization has occurred. In a primary inoculation they may pass through the first glands and find lodgment in the next group or the group still further on, consequently we are at a loss to find when and where they gained access to the tissues.

Infection Through the Respiratory Tract.—For many years it was taken for granted that tuberculosis was an aerogenous infection and that the chief route of infection was through the inspired air. That the lungs were the seat of involvement made this seem certain. However, in recent times, the route of infection has been a subject of wide discussion.

Cornet in producing experimental tuberculosis in animals by causing them to inhale dust containing large numbers of tubercle bacilli confirmed the fact that infection could take place by the aerogenous route and practically established this as the mode of infection for the time being. It was the original belief that the inspired air carried the bacilli directly into the alveoli of the lung; and, in fact, this theory is still held today by many men and is supposedly supported by a great mass of evidence such as that brought forth by E. Albrecht, H. Albrecht and Ghon, who have made thorough examinations of the lungs of children, postmortem, and have shown that whenever the bronchial or peribronchial glands are involved there is a focus, usually small, in that portion of the lung which drains into these enlarged glands. This small focus is taken to be the primary focus, the enlarged gland to be a metastasis. This primary focus in the pulmonary tissue was so commonly found in their researches that they consider that it establishes the aerogenous route of infection beyond doubt. Hamburger accepts the results of their investigations as final. He says, the fact that 95 per cent of all bodies of children who show tuberculous infection have both a focus in the lungs and infected bronchial glands, is sufficient to establish the inhalation theory of infection; and, he believes that the reason the pulmonary focus is so often overlooked is because it is small and overshadowed by the much larger and visible glandular involvement.[8] It seems to me that we must consider the foci described by these observers as being possibly a result of either direct inhalation or an indirect infection through the blood stream, the bacilli entering through the intestinal mucosa, and being carried via the lacteals and thoracic duct. I have never been convinced, however, that bacilli are commonly carried to the alveoli of the lungs by the inspired air. In an early paper[9] when discussing this question I drew attention to the protective cilia in the anterior portion of the nose, the irregular mucous surfaces inside the nose, which are normally covered with moisture; the acts of sneezing called into play when foreign substances are lodged on the mucous surfaces; the fact that the currents of air carrying the bacilli impinge on the moist surfaces of the posterior wall of the oropharynx and that the air must then pass into the lower air channels and again follow a course of angled passages, coming in contact at every turn with moist surfaces, covered with ciliary epithelium, which are waving toward the larynx, before the alveoli are reached.

It would seem far more probable that most bacilli which find their way into the upper air passages are deposited on the mucous membranes and are either expelled or swallowed or find their way through the walls into the lymph spaces to be carried to adjacent glands. The fact that there is no direct air current deep in the lungs would make the direct passage of bacilli into the alveoli difficult, even though they passed into the larger air passages. In most individuals beyond the early years we must assume that cellular defense is present which makes implantation from without extremely difficult.

Comparison of Infection in Tuberculosis and Definite Air-Borne Disease.—We are not in a position, however, absolutely to deny that bacilli gain entrance to the lower air passages, particularly the larger ones, through the inspired air. We must admit that this is probably the way pneumonia, influenza and bronchitis establish themselves, but the specific germs which are accountable for these diseases live in a saprophytic state on the walls of the upper air passages, while tubercle bacilli do not. Their incubation time is short; that of bacilli is long. These diseases primarily affect the upper passages and then extend toward the alveoli. Tuberculosis, on the other hand, as we find it in the lungs, is most apt to start at the apex and travel downward through adjacent lymph spaces or through the adjacent bronchi; so the very localization and subsequent spread of clinical pulmonary tuberculosis as compared with the definitely air-borne diseases combats the idea of direct aerogenous infection.

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[8] Tuberkulose der Kinder, Handbuch der Tuberkulose, Brauer, Schröder und Blumenfeld, Bd. v, 1914.

[9] The Mode of Infection in Tuberculosis and Measures for Its Prevention, Journal of Tuberculosis, vol. v, 1903.

Further than this, careful examination of the air passages of animals fails to support this theory of direct aerogenous infection. While the nose, pharynx and mouth show many bacteria, the lungs of freshly killed animals are wholly or almost wholly free from bacteria. This has been proved by many investigators, among whom are Müller[10] and Bartel.[11] That finely divided dust particles can find their way into the lungs through direct inhalation of atmosphere surcharged with it is possible. We must accept this as the case in anthracosis. We cannot conceive, however, of there being any parallel between the carbon in the city atmosphere or in the coal mines or the dust in the stone quarries and the tubercle bacilli contaminating inspired air. In the former instance the air is surcharged with particles and inhaled over a prolonged period of time, while in the case of tubercle bacilli there are comparatively few in the air and, as a rule, they are not inhaled over a prolonged period of time.

The objection has been made to the experiments of Cornet that he used dust so thoroughly saturated with tubercle bacilli that the experiments were not natural. Further, attention should be called to the fact that in his experiments infection did not occur in the apex of the lung, but in the lower portions which directly receive the inspired air.

Droplet Infection.—Fluegge[12] brought forth the theory of droplet infection. His theory is that it is not so much the dry bacilli, mixed with dust, which are inhaled into the lung with the inspired air, but the fine particles of bacillus-bearing secretion which are thrown out by the patient when coughing and sneezing. These fine particles, according to Fluegge's theory, are suspended in the air and inhaled directly into the lungs, in a moist state before the bacilli have been seriously injured by outside environment. Fluegge has produced tuberculosis in animals experimentally by putting them in a box and having tuberculous patients cough over them. We can say of this experiment, however, the same as of Cornet's, that it proves nothing except that tubercle bacilli can produce tuberculosis in animals under conditions where sufficient bacilli gain entrance to the tissues. The bacilli taken in through droplet infection should be more virulent than those that have dried and passed into dust. The main fact against this theory is that the moist particles of sputum carried in the atmosphere would most likely be deposited on the mucous membranes before they reach the finer air passages of the lung; in fact, it seems more probable that these would be deposited in the oropharynx and swallowed.

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[10] Die Keimgehalt der Luftwege bei gesunden Thieren, Münchener Medicinische Wochenschrift, 1897, Nr. 49.

[11] Über den Bakteriengehalt der Luftwege, Centralblatt für Bakteriologie, 1898, Bd. xxiv, Heft 11 u 12.

[12] Über Luftinfection, Zeitschrift für Hygiene und Infectionskrankheiten, Bd. 25, 1897.

Infection Through the Alimentary Tract.—In considering infection through the alimentary tract we include infection through the tonsils, the mucous membrane of the mouth and oropharynx, as well as the entire lower portion of the gastrointestinal tract.

The theory of infection through the alimentary tract is well established. The ability of bacilli to withstand the action of the gastrointestinal secretions and the putrefaction which takes place in the bowel, likewise the penetrability of the mucous membrane of the intestinal tract has been proved. Bacilli have been found in the tissues of animals within a few hours after their ingestion. The fact is now well established that bacilli prior to the time when a specific defense has been established, pass through mucous membranes without leaving a trace of their passage. It is also well established that they pass through both healthy and diseased mucous membranes.

The experiments of Calmette and Ravenel in feeding non-infected dogs with tubercle bacilli show the readiness with which the mucous membranes are penetrated. Until infection has occurred and resulted in the endowment of the body cells with specific defensive powers, we must consider that little or no barrier is offered to the bacillus by the mucous membranes; at least, no specific antagonism is shown, no special attempt to prevent the bacilli from entering the body. The mucous membranes are more like meshes which permit an easy penetration, the bacilli passing on to be caught in the lymph glands; but, after the cells have developed a power of defense, a specific reaction is offered by the local cells and the mucous membranes themselves become resistant, otherwise how can we account for the resistance of the air passages and intestines in advanced pulmonary tuberculosis?[13]

If the bacilli swallowed by the average advanced tuberculous patient penetrated mucous membranes as readily as they do in the non-infected, there would be no chance for the patient. The infiltration and ulceration of the intestines in advanced tuberculosis must be looked upon as an attempt on the part of the cells to exercise their specific protective powers against the bacillus. The infection is from surface inoculation and the infiltration or ulceration is the result of the inflammation which takes place between the sensitized cells and the bacilli and must be looked upon as an expression of an attempt to eliminate the bacilli instead of allowing them to pass into the deeper tissues.

The writer was early convinced that lymphatic infection in childhood stood in a close relationship to the clinical disease in the adult and discussed this question before the Twenty-ninth Semi-Annual Meeting of the Southern California Medical Association in May, 1902.[14] At that time we did not have the various tuberculin tests which have proved so valuable in studying this disease in childhood. My observations were based on the frequency of glandular tuberculosis found in children postmortem and the clinically enlarged lymphatic glands which were later followed by tuberculosis in the same individual.

The observations of Blos[15] who followed the after history of

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[13] Pottenger: Some of the Anatomic-Pathologic Problems in Tuberculosis, Transactions of the American Climatological Association, 1915.

[14] Tuberculosis of Childhood With Special Reference to Infection, Southern California Practitioner, June, 1902.

[15] Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1899, Nr. iv.

160 cases of tuberculosis of the lymphatic glands from Czerney's clinic in Heidelberg for a period of from three to twelve years and found that 40 per cent developed clinical tuberculosis, were quoted. He states: "Just what per cent of the cases of tuberculosis in adults is due to this lymphatic infection during childhood we are unable to say; yet, it is a source of infection that deserves much more consideration than it has hitherto received."

Von Behring[16] announced the important fact that the intestinal tract of the newly born is especially easily penetrated by bacilli. He further asserted that tuberculosis is a disease which enters the human body through the intestinal tract, that childhood is the time of infection, and that the source of infection is almost wholly milk. While this latter has not been substantiated, childhood as the time of infection and the alimentary canal as the chief portal of entry has been rapidly gaining adherents.

When the idea of alimentary infection was first suggested, those who opposed it put forth the argument against it that the intestine and glands of the mesentery are not usually infected; but later experiments have shown that this is not necessary. Bacilli may pass through the mucous membrane, be taken up by the lymphatics, carried by way of the thoracic duct and poured into the subclavian vein to be strained out in the lung or other organs. It is further possible that infectious material may pass through the mesenteric glands and on into the mediastinal glands the same as bacilli may pass from the tonsils through the superficial cervical down into the deep cervical and possibly on into the mediastinal glands. This latter possibility, however, is not supported by Most's[17] anatomical studies.

Rabinowitsch and Oberwarth[18] injected tubercle bacilli into the rectum of animals and showed that they were taken up and carried into the intestinal glands and that they were also taken into the blood and found in the lungs. From studies of the intestinal tract we know that material injected into the rectum passes rapidly toward the ileo-cecal valve and when patulent even passes beyond it. The above experiment shows that tubercle bacilli may be taken in through the mucous membrane of the intestinal tract and carried directly to the lung, although it does not reveal the point of entry; consequently, *it is possible to have what seems to be a primary infection of the lung take place through the intestinal tract.* This suggests another explanation aside from that of direct inhalation for the cases described by Ghon, the Albrechts and Kuss.

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[16] Tuberkulosebekämpfung, Marburg, 1903.

[17] Die Topographie des Lymphgefässapparates des Menschlichen Körper und ihre Beziehungen zur den Infektionswegen der Tuberkulose, Stuttgart, 1908.

[18] Über die Resorptionsinfektion mit Tuberkelbazillen vom Magendarmkanal, Berlin klinische Wochenschrift, 1908.

The confusion arising from the interpretation of inoculation experiments is due to the fact that the inoculations are carried out in an unnatural way. Much larger doses of bacilli are employed than would be found in normal inoculations, and they are taken into the body in ways that are unnatural; consequently, conclusions that are drawn must be more or less guarded.

In connection with the experiments with bacilli we must mention those of Calmette and others in the production of anthracosis by injecting the soot particles into the alimentary canal. Schultze[19] says in discussing his experiments with the ingestion of India ink: "It is the mediastinal glands which are first and most involved. They show color before the lung tissue itself." And again: "One can say with the greatest certainty that the particles pass through the lymph channels and thoracic duct into the blood stream." These experiments show unnatural conditions, but prove that the path from the intestinal tract to the lungs or bronchial glands is an open one. He further states that he finds the spleen, liver, kidney and other organs to contain small particles as well. This same has been shown by others who have carried on their experiments by inhalation, among whom are Sluka.[20]

Not only must we consider the ingestion of tubercle bacilli along with food or drink, but we must consider the possibility of children infecting themselves through bacilli that are carried to the mouth from their fingers after playing on the floor or handling infected articles. We must also consider the possibility of bacilli, being taken in with inspired air and being deposited on the moist surfaces of the oropharynx, either passing through the mucous membranes or the tonsils or being swallowed and then causing infection through the intestinal wall.

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[19] Intestinaler Ursprung der Lungenanthrakose, Zeitschrift für Tuberkulose, Bd. ix, Heft 5, 1906.

[20] Über die Wanderung korpuskular Elementen im Organism, Prager Medizinische Wochenschrift, 1898.

Nägelsbach examined the dirt taken from under the finger nails of children and found tubercle bacilli present in quite a percentage of the specimens.

Tonsils.—The tonsils have been considered a point of entry for tuberculosis for many years. Some authors such as Grober[21] starting with the idea as suggested by Aufrecht, that in 82 per cent, and according to Naegeli, 90 per cent, and according to Cornet in practically all cases of pulmonary tuberculosis, the primary infection is at the apex, tried to show that there must be some direct relationship between the portal of entry and the apex of the lung. Accordingly Grober made an extensive study in which he endeavored to show that tubercle bacilli are taken into the human body through the tonsils, pass down into the cervical glands and cross over the pleural lake directly into the apex. This, however, has not been generally accepted. Most[22] in his studies of the anatomy of the lymphatic system fails to furnish an anatomical basis for such lymphatic connection as previously mentioned. We cannot deny, however, that the tonsil and other lymphatic tissue in the pharynx bear an important relationship to infection in tuberculosis the same as we now know it in other infections (see Figs. 13 and 14, pages 93 and 94).

Aufrecht made an examination of 10 tonsils in which he found tubercle bacilli twice, and 16 adenoids in which he found tubercle bacilli three times, thus making in this small number 20 per cent in the tonsils and 19 per cent in the adenoids.

Dieulafoy[23] extirpated hypertrophied tonsils and adenoids and inoculated them into guinea pigs. Of 61 hypertrophied tonsils, 5 or 13 per cent caused infection. Of 35 adenoids 7, or 20 per cent produced infection.

Such experiments as these show that tonsillar tissue does contain tubercle bacilli and inasmuch as the histological tubercle is not present we must assume that the bacilli are there in the process of being destroyed or passing through the gland, the same as they pass through the mucous membranes of the air passages and intestinal tract. Their course after passing through the tonsils is first directed into the cervical glands and from there it is just as simple for them to pass into the blood stream and be carried to the apex of the lung as it would be for bacilli to be carried from the peribronchial or glands in any other portion of the body. The entrance of tubercle bacilli or any other microorganisms through the tonsils is favored not only by the character of the tonsillar tissue, but also by the peculiar position which the tonsils occupy in the throat. The same might also be said, only the position is not as favored, of the adenoid tissue and the mucous membranes of the oropharynx in general. The tonsillar tissue in particular has many crypts. There is considerable force exerted in the act of swallowing and this force must press food particles, and, with it, bacteria strongly against the tonsils and the mucous membrane of the oropharynx and in this way favor the passage of bacilli into the tissues. That bacilli may pass through the tonsils and cause infection must not be taken as an argument in favor of the indiscriminate removal of the tonsils.

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[21] Die Tonsillen als Eintrittspforten für Krankheitserreger bezonders für den Tuberkelbazillus, Gustav Fischer, Jena, 1905.

[22] Die Topographie der für die Infektionswege der Lungentuberkulose massgebenden Lymphbahen, VI Internationale Tuberkulosenkonferenz, 1907, Bericht S. 132.

[23] Bulletin de l'academie de medicine, 1895, Nr. 17-20. Cited by Kössel, Zeitschrift fur Hygiene, 1896, Bd. 21, S. 68.

The importance of infection through the alimentary tract has been greatly accentuated by the study of the intertransmissibility of human and bovine tubercle bacilli. If the theory of bovine infection, particularly milk infection, is to hold, the alimentary tract must furnish an important portal of entry. While this question does have a very important bearing upon the question of intertransmissibility, it has a much wider meaning in the general infection of the human body by tubercle bacilli regardless of the source. It is impossible for us to believe that all infection is accounted for by the inhalation of bacillus laden dust and the ingestion of infected milk. That this is true is evident if we study tuberculosis as we find it in inhabitants of different countries. For example, the great amount of tuberculosis in all forms that is present in Japan where comparatively little milk is used, shows that infection through the ingestion of milk is not a necessary factor. Tuberculosis found in the United States where large quantities of milk are used, assumes the same forms as it does in Japan.

In a recent paper, Ravenel,[24] in an interesting discussion of the question of the modes of infection, gives a very valuable historical presentation of the more important studies in infection through the digestive tract. Because of the practical importance of this discussion I quote as follows:

"**Infection Through the Digestive Tract.**—The first recorded cases of infection through the digestive tract are those of Klenke, who in 1846 gave the histories of sixteen children who had been fed on cow's milk, and all of whom showed tuberculosis of the intestines, glands, skin or bones.

"Previous to this, however, Carmichael, in 1810, recorded as his observation that he had frequently seen the mesenteric glands strumous without involvement of the external glands, and this led him to believe that in scrofula the mesenteric glands were first involved. He referred to the frequency of a disease similar to scrofula seen in pigs fed on sour milk, and called attention to the common occurrence of bowel trouble in children at the time of weaning 'too often followed by disease of mesenteric and lymphatic glands.'

"Casper, in 1882, referred to the extent of tuberculosis ('nodular consumption') among the milch cows of Paris, and said, 'It is possible that there is a connection between this phenomenon and the many tuberculous diseases among the children of Paris.'

"Experimental evidence of tuberculous infection in cattle by feeding was given as early as 1868, by Chauveau, who extended his work with corroborative results in 1872 and 1873; and Villemin in 1869 obtained positive results by feeding in rabbits and guinea pigs. He was the first to employ a tube for the introduction of tuberculous material into the stomach. Experiments by scientists of every nation have given positive support to the work of these early observers. It is also a well-known and widely observed fact that in the spread of tuberculosis of cattle and swine the digestive tract plays a most important role. Swine particularly are infected through the digestive tract in the great majority of cases, and the ingestion of milk from tuberculous cattle is universally recognized as one of the chief factors in the spread of the disease among these animals. The first examination made by the officers of the Federal Meat Inspection Service is based on this fact. In 93.3 per cent of cases, tuberculous hogs show infection of the cervical glands.

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[24] Present Views in Respect to Modes and Periods of Infection in Tuberculosis, Journal American Medical Association, February 26, 1916.

" 'As the disease is essentially produced by ingestion, the glands and tissues associated with the digestive tract are the most frequent seats of infection. Indeed, the superior cervical glands (in almost all cases the submaxillary gland) are nearly always affected, as at the postmortem examination held by bureau inspectors over a consecutive period on 120,000 tuberculous hog carcasses, 93.3 per cent were found to contain lesions in these glands. The large tonsils and the large number of lymph sinuses in the lymph glands probably account for this great frequency.'

" 'From some records relative to the locations of tuberculous lesions in cattle that were made at the same time that the figures pertaining to hogs were collected, it appears that in tuberculous cattle the lesions of tuberculosis were located in the cervical glands in 66⅔ per cent of the animals, in the bronchial glands in 54.5 per cent, and in the mediastinal glands in 63.6 per cent.'

"In 1901, in his London address, while discussing the relation of bovine tuberculosis to human death, Koch took the ground that infection through food could be assumed to have taken place with certainty only when the primary tuberculous lesion was located in the intestines. He asserted that this was rarely found to be the case, and argued that the danger of infection through food was slight. The stand taken by Koch assumed that the tubercle bacillus was unable to gain entrance to the system through the intestinal wall without the production of a lesion at the point of entrance.

"The question raised by Koch has been thoroughly investigated, and it can be said with certainty that the tubercle bacillus can penetrate the mucous membrane of the upper digestive tract as well as that of the intestine readily and quickly, without producing a tuberculous lesion at the point of entry. By Baumgarten, however, it is held that there is always a focus of infection at the point of entrance sooner or later, even though it may be microscopic. Against this is the demonstration by von Behring and Roemer of the ready permeability of the intestinal mucosa of young animals by various bacteria—even the large anthrax bacillus. In 1,000 swine, infected through food, Ostertag found the glands of the throat, neck and mesentery tuberculous, while the mucous membrane of the intestine was always free from disease. Orth, in 1879 and Cornet, in 1880, observed bacilli pass step by step through the fully developed mucous membrane of the uninjured intestine and reach the lymph channels and mesenteric glands, leaving no recognizable trace behind (Fürst). Dobroklonski, in 1890, working under Cornil, showed that the tubercle bacillus would quickly penetrate the healthy wall of the intestine in guinea pigs. Desoubry and Porcher, students of Nocard, showed in dogs that during the digestion of fats large numbers of bacteria were carried through the intestinal wall and could be detected in the chyle within a few hours after the meal was given. If food free from fat was given, few or even no bacteria could be found. Nicolas and Descos, in 1902, found that tubercle bacilli given to dogs in fatty food reached the chyle within three hours. During feeding experiments conducted at the laboratory of the State Live Stock Sanitary Board of Pennsylvania, we frequently observed extensive tuberculosis of the lungs and thoracic glands in animals which showed slight or even no involvement of the intestine. In 1902-1903 I introduced into the stomach of a number of dogs tubercle bacilli suspended in an emulsion of melted butter and warm water, using a tube in order to prevent possible infection through the trachea. The dogs were killed after three and one-half to four hours, during active digestion, as much chyle as possible was collected, and the mesenteric glands were removed. Guinea pigs were inoculated with this material. Tubercle bacilli were demonstrated in abundance in eight of ten experiments. The dogs were kept on soft food for some days before the experiments, and were purged with castor oil, in order to rid the intestine of all foreign matter which might injure the mucous membrane. Numerous sections of the intestine were examined also, but no injury could be detected.

"It is possible here only to mention the work of Calmette and his students, as a result of which he concluded that pulmonary tuberculosis acquired at any age may be due to recent intestinal infection.

"The rapidity with which tubercle bacilli penetrate the intestine and reach the lung through the thoracic duct is remarkable. Besanti and Panisset found that when fed to dogs in soup they reached the heart blood within from four to five hours. Bartel found that after a single dose they penetrated the uninjured intestine and reached the mesenteric glands during the following digestive period. Schlossmann and Engel, by inoculating tubercle bacilli suspended in cream into the stomachs of young guinea pigs through an incision in the abdominal wall, demonstrated that they reached the lung within six hours. Their work has been confirmed by Ravenel and Reichel. Rabinowitsch and Oberwarth established nutrition through a gastric fistula in swine, and then resected the esophagus, after which tubercle bacilli were introduced into the stomach. Within twenty-four hours they were shown to have penetrated to many organs of the body."

Difficulty in Determining Source of Infection.—Jacob in his exhaustive study of the children in the country districts of Germany, shows that tuberculosis is very prevalent and found in families where there have been no open cases of tuberculosis for many years, among children who do not make milk their principal article of food; so it seems to me that we must not look for other sources of infection and other routes of bacilli so much as for other methods of carrying the bacillus and other bacillus carrying material.

Jacob suggests the fly as a carrier and is of the opinion that it is a big factor. Here, however, we must remember that flies, as a rule, do not go far from the place of their birth, consequently they will not carry the disease to any great distance. Is it not possible that one source of infection in tuberculosis is articles of food that are prepared and handled by tuberculous people, or food that is exposed to dust that contains tubercle bacilli. Further, is it not possible that in the handling of wares of different kinds that tubercle bacilli are carried into the homes in sufficient amounts to infect children? The infecting bacillus is so ubiquitous that we must recognize the fact that it is brought to the body in many different ways, and not be content by thinking that it is simply inhaled through dust and ingested with milk; otherwise, how can we account for the numbers of infected children who seem to be removed from these more common sources of infection?

Jacob examined the flies in nineteen peasant's houses and found tubercle bacilli on the feet in 6 or 31.5 per cent. This is sufficient to condemn the fly and call for its extermination.

Jacob examined 2,744 children in 27 villages in the country districts of Huemmling. Of these, 920 reacted positively to tuberculin and 1,829 proved negative. The 920 children who showed a positive reaction, lived in 620 houses. Among these were 237—38.2 per cent of houses in which during the last decade one or several members of the family had died or suffered from tuberculosis. The remaining 383 houses, equal to 61.8 per cent had previously shown no cases of tuberculosis. The 1,829 children reacting negatively lived in 526 houses. Of these 154—29.4 had housed tuberculous patients and 372 or 70.6 per cent of the houses had not contained a single case of tuberculosis.

In these statistics it will be seen that 383, or 61.8 per cent of the 620 houses in which these children who showed a positive reaction, lived, had not housed patients suffering from tuberculosis.

He also examined children who were not going to school in villages which had been free from tuberculosis and found that from 30 to 40 per cent of the children were infected. Jacob excludes milk as being the principal factor in this infection, for he says that in these districts the children are mostly breast fed and are often nursed during the entire period of the first two years of life; and, he calls attention to the fact that owing to the care which the mothers give their children and the advantage of breast feeding, the mortality among these children is exceedingly low. Of 600 children born each year, the mortality is only 11.7 per cent the first year. He then compares this low death rate with the death rate for Bavaria in the year 1909. In Oberfranken the mortality was 15 to 20 per cent, in Mittlefranken it was 24 to 30 per cent, and in Schwaben and Neiderbayern it was 26 to 30 per cent.

These statistics of Jacob are of great importance in their bearing upon the method of carrying infection. Here we have a large body of children who were free from contact with tuberculous individuals, who have never lived in homes occupied by tuberculous patients, who were fed on breast milk until the second year and in whom the general mortality is much lower than that usual among children; yet a large percentage of them show infection before the school age.

Other Methods of Infection.—While infection through the respiratory and gastrointestinal tracts will most probably account for nearly all cases of tuberculosis, yet it is necessary to mention that tubercle bacilli may be taken in through the skin. This at times we see in local tuberculosis of the fingers and hands of those who perform many postmortems on tuberculous subjects, also on the hands of those who handle tuberculous meat. It is also possible that infection might take place through other mucous membranes such as those of the genitourinary tract; but such infections are comparatively rare.

No matter how the bacillus is taken into the body, unless destroyed, it follows one of two courses after it gains access to the tissues. It either forms the tuberculous focus where it settles; or, which is far more common, it passes into the lymphatic spaces and is carried to the neighboring glands or to other glands where it forms a primary focus; or it is taken up, as it sometimes is in the case of the gastrointestinal tract, by the lymphatics and carried to the thoracic duct and poured directly into the blood stream.

The spreading of the disease from the primary focus in the lymph glands takes place either by bacilli penetrating the walls of blood vessels and being carried to the lungs or other parts of the body, or through the wandering cells; or directly through the lymph itself. In this connection it must be remembered that infection may follow the back flow of lymph as well as the onward flow (Tendeloo). It is possible that the adaptability of the tissues to infection and to the particular strain of bacilli (Rosenow) are factors in determining the secondary localization of the bacilli. In the human race we find primary tuberculosis in the glands and metastatic infection most usually in the lungs of adults, while in rabbits the lungs and kidneys are usually involved and in guinea pigs very often the spleen is involved. With reference to these infections in animals we must bear in mind that they are infections on virgin soil, in which specific cellular defense has not been developed, consequently, there is not this specific inflammatory reaction which takes place between bacilli and specific enzymes to prevent widespread dissemination.[25]

Chronicity depends on the invading bacilli having a relatively low virulence. This property may be either in the germs themselves or it may consist of marked defensive characteristics; those which are naturally possessed by the host or which have been acquired naturally or through previous infection.

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[25] Pottenger: Tuberculin Therapy—Its Present Imperfections and Future Improvements, Medical Record, February 20, 1915.

CHAPTER III.

RELATIONSHIP OF THE PRIMARY FOCUS TO CLINICAL TUBERCULOSIS.

Differentiation Between Primary Focus and Primary Metastasis.—In the following discussion I shall speak of the primary focus, meaning the first area of infection which takes place in the body; and of the primary metastasis, meaning an area of infection which occurs in some part of the body, the bacilli causing the same having come from the primary focus. Secondary metastases are those which are formed by bacilli which escape from a previous metastasis.

It will be seen at once that the primary focus is an infection in a previously uninfected organism, while metastases occur in organisms which have already been infected. These two types of infection are very different; the former occurs without the opposition of specific defense and the latter is opposed by the specific reaction which has been developed by the body cells. In comparing the phenomena which are associated with primary infection with those associated with metastatic infection, particularly the phenomena which occur in connection with the lymph glands which receive the drainage from the tissues affected, the same relationship is apparent as is noted between the seat of inoculation and the regional lymph glands in Koch's inoculation experiments which have been quoted so much in our recent studies of hypersensibility. If the same principles hold, and I see no reason why they should not, we can determine whether or not a lesion in the lung is a primary, or a secondary, or metastatic infection by investigating the manner in which the glands behave toward the focus of infection.

It will be recalled that Koch's experiment shows that the inoculation of a previously healthy animal with tubercle bacilli produces very little disturbance at the point of inoculation; but the bacilli are taken up and at once carried to the regional lymph

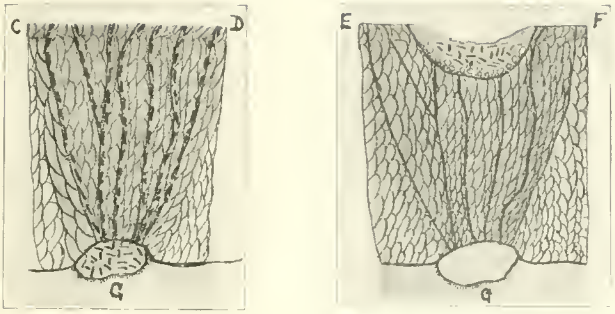

Fig. 12*A*.

Fig. 12*B*.

Fig. 12, *A* and *B*.—Illustrating schematically the difference in behavior of tissues toward invading bacilli. Fig. 12*A*, when specific defense has not been established by previous infection; Fig. 12*B*, when specific defense has been established by previous infection. In Fig. 12*A* the bacilli pass through the mucous membrane *C-D* and the tissues beyond and are carried directly into the lymphatic gland *G*. In Fig. 12*B*, they do not pass through the mucous membrane *E-F* readily, but are held at the point of implantation. The tissues between the mucous membrane and the gland *G*, as well as the gland itself, are free from invading bacilli.

glands where a marked infection is established. However, after infection has once been established in the body, the behavior of the tissues is very different. They are now on guard against tubercle bacilli and attempt to destroy them at the site of inoculation and prevent their implantation. This condition we describe as sensitization of the body cells. It means a condition in which the cells produce a specific reaction. Through the original infection all body cells have been changed so that they now offer a specific protection against any new bacilli which attempt to enter the tissues; consequently there is produced in an animal which is already tuberculous, when inoculated a second time, an induration at the point of inoculation followed sooner or later by ulceration, the regional lymph glands not being involved or being involved only slightly. Figs. 12*A* and 12*B* illustrate schematically the difference in behavior between bacilli and the tissues prior to and after the development of specific cellular defense. Specific defense is due to specific enzymes.

In the primary pulmonary foci described by Ghon, he calls attention to the insignificance of the pulmonary lesion and the manner in which it is overshadowed by the marked swelling of the regional bronchial glands which receive drainage from the pulmonary tissue involved. This is markedly different from the metastatic foci which occur in the lung later in life in which the swelling of the lymph glands plays such a minor role. Often even in advanced, widespread, and even markedly active tuberculosis of the lungs, we find a very insignificant involvement of the peribronchial lymph glands, although they have often been subject to drainage for months and sometimes for years. *So, in primary infection there is a slight lesion in the tissues of the organ involved and a marked infection of the regional glands, while in metastatic or secondary lesions, the tissues of the organs bear the brunt of the infection, while the glands remain relatively free.*

We see the same thing in the intestinal tract. A secondary infection in the intestinal tract may produce a widespread ulceration of the gut with very slight or no tendency to involvement of the regional lymphatic glands. The same is true in the larynx. A secondary involvement of the larynx is not followed by a marked disturbance on the part of the regional lymph glands which receive drainage from it, even though the process be extensive and very active.

These facts help to explain the difference in the behavior of tubercle bacilli when taken into the body under different conditions. When an infection has once occurred, thereafter there is an attempt on the part of the tissues to limit a new inoculation to the point of entry while a primary infection is carried rapidly to the lymphatic glands. According to this we can more readily understand the tendency which appears on the part of secondary metastases in the lung to go on to cavity formation. It is due in part to a specific reaction of the cells by which they put up a specific local defense and make an effort to eliminate the infection, the same as the ulceration which takes place at the point of inoculation in Koch's original experiment: so, we see a parallel in primary infection of the lung with marked secondary involvement of the lymph glands to the inoculation of healthy guinea pigs and the marked glandular infection; likewise in metastases in the lung with comparative freedom of lymph gland involvement to the inoculation of the guinea pig which is already tuberculous, where local ulceration forms and the lymphatics remain free.

Tuberculosis is Primarily a Lymphatic Disease.—Primary foci, such as those described by Ghon, are comparatively so insignificant that we are justified in saying that tuberculosis is primarily a disease of the lymphatic system. Following infection with tubercle bacilli, the lymphatic glands may enlarge and become the seat of active tuberculosis; the disease may heal; or it may remain quiescent without healing. Other portions of the body may be infected from this primary lymphatic focus either by bacilli passing out into the lymphatic spaces or by being carried by wandering leucocytes or, which is more common, by their passing through the walls of blood vessels which are involved in the inflamed tubercle. If the bacilli in the glands multiply and the focus undergoes necrosis, the small blood vessels adjoining it, or entering the tubercle, partake of the inflammation; and, at times, bacilli pass through the vessel walls and are carried to distant portions of the body.

Metastatic Tuberculosis.—During early life the bacilli escaping from the lymphatic glands may be implanted in any portion of the body, organs other than the lungs being very commonly affected. In later life after adolescence, the lung, particularly the apex, is the most common seat of metastases. The primary metastases which first form in the tissues after bacilli escape from the lymphatic glands are, as a rule, small and non-virulent in character. As mentioned elsewhere, this is probably due to several factors. The fact that bacilli escape into the blood stream and are diluted causes the infection to be produced by few bacilli. While circulating in the blood the bacilli are acted upon by the antibacillary elements found therein which have a tendency to reduce their virulence. Those in clumps are most apt to escape this action. Aside from this there is the protective influence of the cells at the point of implantation which are endowed with specific defense and which must be overcome before infection can take place.

That this primary metastasis often heals we are led to infer from the scars that are found in various portions of the body, particularly the lungs, postmortem. Where it does not heal it may remain quiescent for either a short or long period of time and then take upon itself activity and produce clinical symptoms.

Whether this primary metastasis shall be accompanied by clinical symptoms or not depends upon the localization. In such a delicate tissue as the meninges, a small focus is sufficient to cause symptoms. Likewise, in a joint, clinical symptoms might be produced by a very small focus. In the bone, symptoms would probably appear somewhat later. In the kidney, liver, or spleen, foci could exist for a long time without symptoms appearing. So may they in the lung.

Relationship of Primary Metastasis to Clinical Tuberculosis.—In order to understand pulmonary tuberculosis as we find it, it is important to recognize its relationship to this primary metastatic focus. We are prone to speak of tuberculosis of the lung being typical tuberculosis and speak of tuberculosis of other organs as being atypical; when, in reality, we are probably comparing the primary metastasis in such tissues as the meninges, joints, and glands with extensions of the process (secondary metastases) in the lung. There are often years between the time of the formation of the primary metastasis in the lung and the onset of symptoms of clinical tuberculosis, while in the more sensitive organs as the meninges and joints, particularly, clinical symptoms follow quickly after the metastasis forms, as previously mentioned.

Clinical symptoms do not appear on the part of the lung until such time as the bacilli, which have been shut up in this primary metastatic focus begin to multiply and give off toxins, which produce changes in the surrounding tissues; and, until bacilli escape into these tissues, producing secondary metastases. With the multiplication of bacilli, toxins are produced and many of the bacilli themselves go into solution. The tissues are injured and necrosis of tissue often occurs. The various products resulting from these processes diffuse further into adjacent tissues producing more widespread inflammatory change. The character and extent of the resultant inflammation depends upon the virulence of the infection, which is equivalent to saying upon the rapidity with which the bacilli multiply and the amount of toxins produced. It may be very slight. Under such conditions the irritation in the tissues is of slight degree. The tissues are also subject to exudation. This may be serous, fibrinous, sanguinous, cellular, or purulent. Further, the tissues that are subject to this collateral inflammation may undergo any of the changes which the tubercle bacillus itself may undergo. They may be organized and produce new tissue or they may go on to necrosis and destruction of tissue.

The most serious aspect of this collateral inflammation, however, is that it works injury to the tissues and produces a stagnation of lymph and forms a ready nidus for further metastases. Bacilli escape more or less frequently from the primary metastasis into adjoining tissues and there find lodgment. The new or secondary metastasis is very apt to be governed, considerably, in its degree of virulence by the original one. If the amount of irritation produced by it is slight and the change in the tubercle itself conservative, not only is the collateral inflammation of a mild degree but few bacilli only are apt to find their way out of the original focus to establish new metastases. In this way we have a mild inflammation of the lung, producing a fibroid or indurative form of tuberculosis. On the other hand, if the original focus is the seat of a very marked activity and is accompanied by severe collateral inflammation more or less rapid necrosis occurs, many bacilli make their escape into adjacent tissues and virulent secondary metastases follow.

In pulmonary tuberculosis it is not until the establishment of collateral inflammation and the formation of secondary metastases that we have clinical symptoms, consequently, what we are in the habit of diagnosing as early clinical pulmonary tuberculosis has as its pathology advanced changes and the production of secondary foci in the lung.

The spread of tuberculosis in the lung is favored by necrosis and caseation of tubercles permitting bacilli to escape from their original focus, by the injury of the surrounding tissue which is produced by the diffusion of toxins with their accompanying exudative inflammation; and, by conditions which favor implantation of bacilli. Necrosis is not necessary, however, for a widespread chronic indurative tuberculosis is often found without necrosis having taken place.

Implantation of bacilli, which escape into adjacent lymph spaces, is favored by lack of motion on the part of the pulmonary tissue, while the implantation of those which escape into the bronchi is favored by motion or coughing.

The primary metastasis in tuberculosis of the lung in the adult is nearly always near the apex. From there the disease extends toward the base either through adjacent lymph spaces or through infection carried by way of the bronchi. In the latter instance sometimes a medium size bronchus will be plugged with mucous and the entire area supplied by it be involved in a secondary infection.

The importance of previous glandular infection in its suggested relationship to adult tuberculosis is borne out by the report of Mönckeberg,[1] who reports postmortems made on soldiers dying from acute diseases and injuries received in battle in whom both active and quiescent lesions were found.

In 85 bodies examined, tuberculosis was found 27 times, or in 31.76 per cent. He included as tuberculosis only cases where definite macroscopic induration was found and excluded all scars which might be questionable or which might prove to be tuberculosis upon microscopic search.

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[1] Professor G. Mönckeberg: Tuberkulose Befunde bei Obductionen von Kombattanten, Zeitschrift für Tuberkulose, Bd. 24, Heft 1, 1915.

These soldiers were strong, healthy men, capable of enduring the hardships of war. They were between the ages of twenty-one and forty-three. Tuberculosis had not been suspected prior to their taking up active duty or they would have been excluded.

Tuberculosis was the cause of death in five cases. It is probable that the activity which went on to the death of the patient was due to lowered resistance incident to the strain of service. In twenty-two cases the tuberculosis was not suspected until found postmortem, although the process was active in three of these cases. In nineteen the process was quiescent. The localization of the lesions is interesting and shown in the table below. It is interesting to speculate on how many more of these foci would have been active had the men been subjected to longer strain.

LOCALIZATION OF UNSUSPECTED TUBERCULOUS LESIONS IN NINETEEN BODIES OF SOLDIERS.

Induration in both apices	3 cases
Induration in the right apex	1 case
Induration in the right lower lobe	1 case
Caseation or calcification of bronchial gland (left)	1 case
Caseation or calcification of bronchial gland (right)	9 cases
(One of these cases also had areas in both apices)	
Caseation and calcification of bifurcation glands	2 cases
(One of these also had calcification of the bronchial glands on the right)	
Caseation and calcification of the glands of the mesentery	4 cases

Leschke[2] calls attention to the marked increase in tuberculosis in the army during the present war as compared with the very low morbidity found in times of peace. During active service tuberculosis has increased markedly. He attributes this difference to the strain incident to the changed condition of life and draws the conclusion which seems but natural, that this fact of itself is strong evidence in favor of the theory that active adult tuberculosis is an extension from an already existing focus, most probably such as are found in the body in childhood.

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[2] E. Leschke: Die Tuberkulose im Kriege, Münchener Medicinsche Wochenschrift, 1915, No. 11, p. 363.

Infections From Without in Later Life.—While we cannot deny the possibility of infections occurring from without the body in later life, yet, according to our newer knowledge, we must recognize that such infections are probably comparatively rare. The early period of life, with its absence of specific cell defense, offers a fairly easy entrance for bacilli, while the period after this specific defense has been established is one of increased resistance. While bacilli, entering the body as they do for the most part in small numbers, may pass through the mucous membranes and be destroyed, without infection occurring; yet, now and then, larger numbers enter; and these, escaping the action of the general agencies of defense, find a nidus in the lymph glands and produce a tuberculous focus. This destruction of bacilli in early life within the body, as well as this early focus of infection, creates the specific cell defense which offers a barrier to the future entrance of bacilli into the body. This specific defense is probably a general property of body cells, as evidenced by the fact that all tested body cells, whether of the skin or mucous membranes, respond with a reaction to tuberculin; and tuberculous foci, wherever found, show the focal reaction to the specific bacillary products.

The effect of this defense is to ward off bacilli and make their implantation difficult or impossible. To what extent it is effective may be judged by the illustration furnished by advanced tuberculosis, which I have mentioned so often. While a few bacilli passing over a mucous membrane gain entrance and cause infection in the non-tuberculous, millions may pass over the mucous membranes daily, after infection has occurred and a specific defense has been developed on the part of the cells, without implantation occurring. This defense offered by the cells of the air passage and intestinal tract, demonstrates that, for an infection to occur, in one who has developed immunity from a previous tuberculous focus, it is necessary that conditions for implantation must be extremely favorable. The quantity of bacilli must be large, they must be held in contact with the tissues for a sufficient time, or the cellular defense must be reduced or in abeyance. Clumps of bacilli, particularly, favor implantation.

We do not know much about the disappearance or reduction of specific defense in actual life, but we assume that it is subject to great variation. Immunity to tuberculosis, as far as we know, is relative; and we believe that it is impossible to develop it to so high a degree that it cannot be overcome in case bacilli should gain access to the tissues in sufficient numbers and be held *in situ* sufficiently long.

It can readily be seen that an entrance through mucous membranes, such as occurs in the natural course of life, and which is so common during the early years, where bacilli enter the body through air or food, would not be easy of accomplishment after the cells have been endowed with specific protective powers, and, could probably take place only in case the bacilli were aided in some such manner as by collecting at some point on the mucous membrane and remaining in contact for a period sufficiently long for the local defense to be overcome to such an extent that the mucous membrane becomes penetrated. That this element of time contact is important, is suggested by the surface infections which we find as complications in advanced tuberculosis. Bacillus bearing sputum passes from the ulceration in the apex of the lung and rarely produces infection along its path except in the last weeks of life, unless through aspiration a bronchus is plugged and the sputum is kept in close contact long enough for implantation to occur. The larynx offers excellent opportunity for implantation because of the fact that the secretions collect in passing the chink; yet, in spite of this splendid opportunity, quite a proportion of patients will go down to death from pulmonary tuberculosis without laryngeal infections occurring. The same is true of the intestinal tract. Infection is most apt to occur in the lower end of the ileum and the cecum and ascending colon where retardation of the contents occurs.

This evidence goes to show that if our specific defense is well developed, surface infection is not easy of accomplishment. Consequently, the conclusion may be drawn that infection through the ordinary paths which succeed in causing implantations in the child is accomplished with difficulty in the adult as long as he is protected by the cellular defense which results from his early infection; and that adult infections, from without, are probably confined to those individuals who have escaped infection in earlier life, if there be such; or those in whom specific defense was poorly developed or from whom it has gradually disappeared; or to instances where bacilli are brought in contact with the mucous membranes from without in large numbers and kept in contact for a time sufficiently long for infection to occur.

The conditions surrounding metastases which take place from the focus within are different. Here the bacilli are already within the tissues. When they make their escape it is into the lymph channels or blood stream, consequently, if they are permitted to remain *in situ* in any part of the body long enough they have exceptional opportunity for causing infection. We are probably warranted, however, in assuming that there are many instances of bacilli gaining access to the lymph and blood stream for every infection which occurs, and that clumping favors implantation. The fact that these secondary metastases are usually of low virulence and the lesion fibroid in form at first, shows the influence of the specific defense upon the microorganisms. These secondary metastases are formed with difficulty and require special anatomical conditions which favor the retention of bacilli *in situ* for implantation to occur. When we consider the relative ease for such implantation to occur through the lymph and blood streams and the relative difficulty of entrance through the surface of the mucous membranes, and consider that the nidus from which the bacilli may come is present in so large a number of adults as a remnant of their early childhood infection, we are justified in considering that the tuberculous disease in the adult is most probably, largely, a result of an infection from within the patient's own body, and not a new implantation of bacilli from without.

The fact that infection often occurs in several members of the same family, one after the other, or at the same time, is brought forth as an argument against this, and as proving an infection immediately prior to the development of the clinical disease. But this is just as likely due to the fact that all were subjected to the same early infections and the same influences which tended to lower resistance and favor activity. Where one clinical case follows another, the depression caused by the disease in another member of the family, or the deprivations caused by the extra tax upon the family's resources, are undoubtedly factors. This factor of extra strain is shown in the case of the soldiers above mentioned.

The size of the early infection is also very important. All else being equal, the larger the early lesion, the greater the danger of future active disease. This explains why so many cases of active tuberculosis occur in families where open tuberculosis existed during the children's early years. The intimate, prolonged association provides the opportunity for massive infection.

This question has an extremely practical bearing in the prevention of the spread of tuberculosis. It shows that children should be shielded as much as possible from infection, particularly massive infection, if we would limit active tuberculosis in the adult. It further suggests that there is no danger in a casual association with the tuberculous; and that, where proper care is used for the destruction of bacillus-bearing discharges, and proper hygienic conditions are maintained, even a prolonged association on the part of those who have developed their specific cellular defense, is without danger. This is borne out by the experience of sanatoria. I have not had a single instance of direct infection of employees and attendants in sanatoria brought to my attention. This is extremely important because of its influence on the future hospital status of tuberculous patients. Phthisiophobia has been so thoroughly inoculated in the mind that in some quarters tuberculous patients have been wrongly refused entrance to hospitals lest they should infect other inmates.

The relationship of previous infections to clinical disease has been shown nowhere better than in our recent influenza epidemic where old quiescent foci were reactivated by the acute infection. The large number of cases of active disease in the lower lobes particularly indicates that influenza had a reactivating influence on quiescent foci.

CHAPTER IV.

TUBERCULOSIS IN CHILDHOOD.

The Natural Defense of the Little Child.—Precedent to a discussion of tuberculosis in childhood, it is well to consider the natural protective mechanism of the child which prevents it from being destroyed by the invading bacillus before it develops an immunity or specific resistance against it.

The natural defense is both humeral and cellular. The cells which take part in the destruction of invading microorganisms belong to various classes. This function is possessed by the endothelial and connective tissue cells, but is strongest in the leucocytes, and particularly the lymphocytes.

Of all structures and systems of the body the lymphatic system plays the greatest protective role. In fact, it is upon this system that the child depends almost wholly for his protection during his early years. It is during this period also that the lymphatic system is most active.

In early childhood the lymphatic tissue is prominent. An abundance of it is placed as an outer defense to guard the vulnerable points of the body such as the vault of the pharynx which meets bacteria which are taken in through inhalation; the root of the tongue and sides of the fauces which are particularly exposed to the bacteria which enter the body by way of the mouth; and in the lower ileum which is particularly exposed to infection by the bacteria which have been ingested, because of the fact that this is a point of stagnation of intestinal contents and rapid absorption.

In case bacteria pass these outer defenses, a second line of defense is arranged in the lymphatic glands which are so situated that they receive drainage from every tissue of the body; and which are located in great abundance near the specially exposed surfaces as exemplified in the cervical, mediastinal, and mesenteric glands. These masses of lymphatic tissue, represented by the pharyngeal, lingual and faucial tonsils and by Pyer's patches as well as the lymphatic glands have the special function of forming lymphocytes and are better provided with the cells upon which the little child must depend for his protection, than any other tissue of the body.

As the child grows older and his body cells, generally, take upon themselves the function of producing specific proteolytic enzymes for his protection, the lymphatic protection which has served him during his early years is no longer so necessary, consequently the lymphatic tissue atrophies.

The importance of the lymphatic tissue in the protection of the little child should be appreciated. The part played by the tonsillar tissue in Waldeyer's ring in warding off infection or failing this, in receiving the infectious microorganisms into tissue which is best prepared for limiting their action, should be emphasized. The tonsillar tissue should not be sacrificed unnecessarily, and it should be the aim of the physician to save its protective function to the child until he has come in contact with bacilli and other microorganisms in sufficient numbers to cause his cells generally to take upon themselves the function of producing specific defensive enzymes. After that has taken place, lymphatic protection assumes a secondary role, and the tonsillar tissue is not so necessary. Figs. 13 and 14 show the natural drainage of the tissues of the tonsils and oropharynx into the deep cervical lymphatic glands and Fig. 15 shows the peribronchial and peritracheal glands in their relation to the bifurcation of the trachea.

Tonsillar tissue which is the seat of dangerous focal infections, or which is interfering with the drainage of the nasal cavities, or threatening the integrity of the ear, or in other ways threatening harm will have to be sacrificed. Where no such cause for removal exists, however, the child should be given the benefit of their protection during his earliest years. They should not be sacrificed uselessly, as though they were of no value.

Infection and Immunity.—Tuberculosis is primarily an infection of childhood. According to our conception, the struggle between the child and the tubercle bacillus begins practically at birth, although the first two years show comparatively little in-

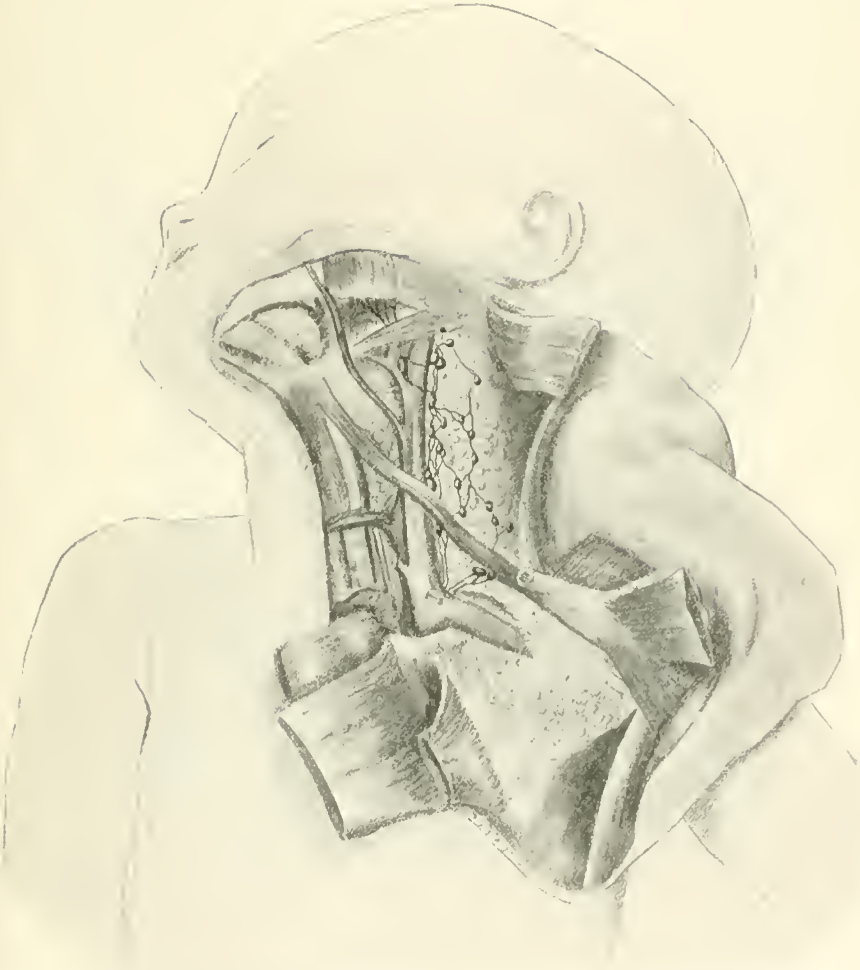

Fig. 13.—Showing the course of the lymphatics from the tonsillar region into the deep cervical glands. (Most.)

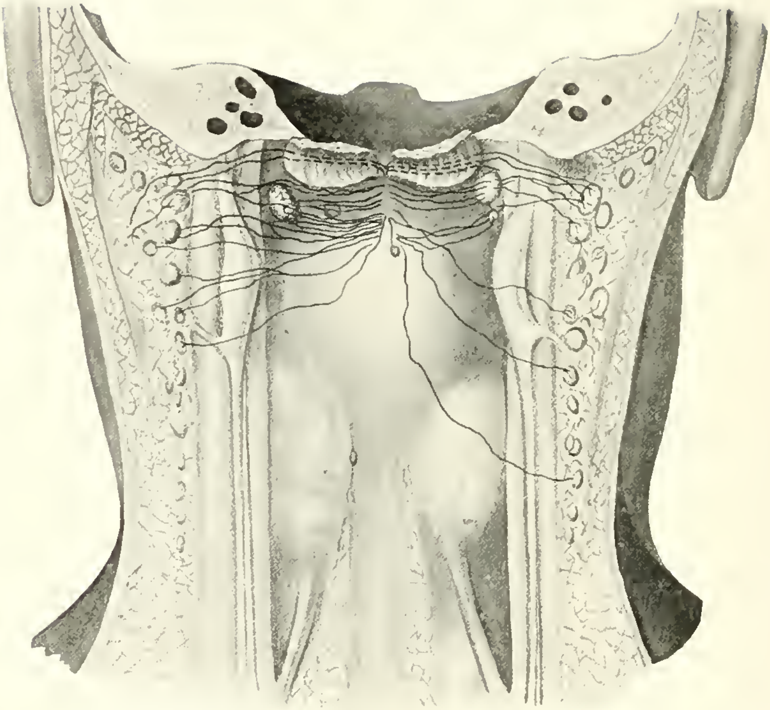

Fig. 14.—Showing the drainage from the oropharynx into the deep cervical glands. (Most.)

fection. This struggle goes on until the resisting power of nearly every child is overcome by the tubercle bacillus to the extent that infection is established.

Coincident with infection goes another process, that of the production of specific immunity. The child takes a few bacilli into the body and they are destroyed. Being destroyed their bodies go into solution. The specific products derived from the bodies of the tubercle bacillus are set free. Specific products are also given out into the tissues from the focus of infection when it has been established. These bacillary substances stimulate the body cells and make them sensitive to further inoculations with tubercle bacilli or their products and cause them to produce specific protective enzymes which both remain attached to the cells and are cast off into the circulation. The result is that in this way a gradually increasing, specific resisting power is built up until the

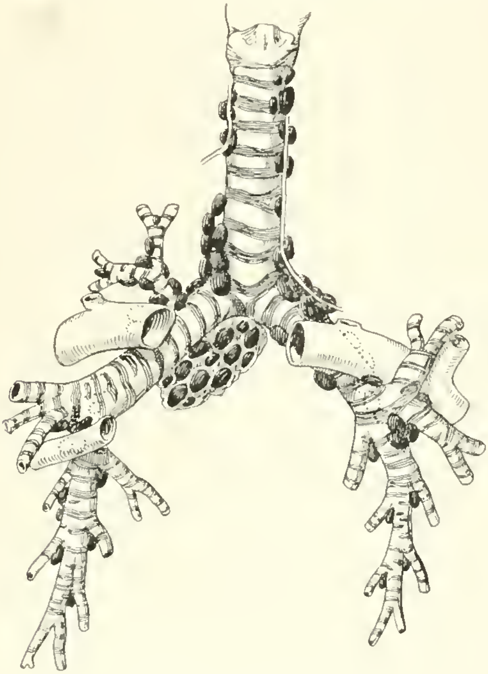

Fig. 15.—Showing the peribronchial and peritracheal glands. (Sukiennikow.)

individual is able to withstand large doses of bacilli, while large numbers at first would be fatal.

In this connection the animal experiments of Webb and Williams[1] are extremely interesting. By the systematic inoculation of guinea pigs with increasing numbers of tubercle bacilli Webb has been able to produce such a state of immunity in the animal that it is capable of withstanding as much as 150,000 tubercle bacilli at a single inoculation without infection occurring.

In speaking of immunity in tuberculosis we must bear in mind that we are speaking of a relative immunity, not an absolute one. While these infections produce sufficient protection to overcome ordinary inoculations of tubercle bacilli, yet it is probable that no individual is absolutely protected from further infection. If the inoculation consists of a sufficient number of tubercle bacilli, or if repeated inoculations, sufficiently large, take place, a new infection will most likely result.

The Difference in the Tuberculous Process at Different Age Periods.—When the child is born he has within his body substances and tissues which protect him against various infections. We must look upon these as being of a general instead of specific nature and not affording a high degree of protection yet sufficient to protect against repeated inoculations of small numbers of bacteria. Sooner or later, however, this natural defense is broken down and the child becomes infected with disease organisms of various kinds. Nearly every child will eventually have one or more of the common infections of childhood. When the child's tissues come in contact with specific microorganisms, as occurs when they are taken into the body or when it overcomes an infection, there is built up within his body a specific defense, a specific immunity against the organism which produces that particular disease. In tuberculosis, the child gradually develops a specific resisting power to tubercle bacilli as mentioned above. This immunity attains such a high degree in advanced tuberculosis, particularly in adult life, that an individual may have millions of bacilli within his body and may be pouring them out in great numbers through the air passages; or, after swallowing them, they may pass through the intestinal tract, and yet his specific resistance may prevent implantation from taking place as previously mentioned.

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[1] VI International Congress on Tuberculosis, vol. i, part 1, p. 174.

Owing to the fact that there is very little specific immunity or resistance against tubercle bacilli during the first years of life, if an infection occurs at this time, it is very apt to be severe in character and, if of large numbers, usually fatal.

This is shown well in statistics arranged in Table I from Hamburger:[2]

TABLE I.

Mortality of Tuberculosis by Age Periods

	I			II	III	IV	V	VI
	1 year			2 years	3 to 4 years	5 to 6 years	7 to 10 years	11 to 14 years
	0 to 3 months	4 to 6 months	7 to 12 months					
		49						
Number of cases..	4	13	32	74	102	38	41	31
		44						
Fatal cases.......	4	13	27	51	69	23	28	16
		90%						
Percentage of fatality........	100%	100%	80%	70%	67%	60%	68%	50%

From this it will be seen that during the first year of life nearly 90 per cent of the cases of tuberculosis are fatal while at the age period of 11 to 14 years such a degree of resistance has been attained that only 50 per cent are fatal.

Hamburger also examined these patients with reference to signs of healing, the results of which are set forth in Table II.

TABLE II.

Frequency of Healed Tuberculosis by Age Periods

	1 year	2 years	3 to 4 years	5 to 6 years	7 to 10 years	11 to 14 years
Total number of cases...........	49	74	102	38	41	31
Number with signs of healing.....	0	0	7	4	7	10
Percentage showing healing......	0	0	7	10	17	33

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[2] Kindertuberkulose, Leipzig and Wien, 1910.

From Table II it is seen that during the first and second years of life there are no signs of healing present. During the third and fourth years signs of healing appear in 7 per cent; during the fifth year in 10 per cent; from the seventh to the tenth year 17 per cent; and, from the eleventh to the fourteenth year, 33 per cent. This is explained by the fact that the child is gradually developing a greater specific immunity, although there are probably other elements which enter and should be considered. Not only is there a difference in mortality from tuberculosis during the early and late periods of childhood, but there is also a great difference in its localization, as will be seen from Table III, taken from Newsholme:[3]

TABLE III.

DEATH RATE FROM TUBERCULOSIS PER 100,000 PERSONS LIVING AT EACH AGE PERIOD

	0-5	5-10	10-15	15-20	20 AND UPWARDS
Pulmonary tuberculosis........	31	20	41	90	176
Tuberculosis meningitis........	109	27	12	6	2
Tabes mesenterica	125	10	7	5	3

The statistics shown for England and Wales in Table III are somewhat different from those on the Continent in that the percentage of tabes mesenterica is much greater in England and Wales than it is on the Continent. It will be noted that the early years are those of non-pulmonary involvement, while the later ones are almost wholly pulmonary. We must conceive of the child as lacking in specific defense; so there is nothing to limit infection when bacilli gain access to the tissues except the natural elements. As a result, the bacilli, if not destroyed, scatter more widely than they do in adults, affecting many organs.

Further emphasizing the frequency of non-pulmonary tuberculosis in young children I desire to call attention to Table IV from Hamburger. In 110 children with fatal tuberculosis, the death in eighty cases was caused by miliary and meningeal tuberculosis, while in only thirty was it due to chronic, general or pulmonary tuberculosis.

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[3] The Prevention of Tuberculosis, 2nd ed., London, 1910.

TABLE IV.

RELATIVE FREQUENCY OF ACTIVE AND CHRONIC TUBERCULOSIS BY AGE PERIODS

CAUSE OF DEATH	I	II	III	IV	V	VI
	1 YEAR	2 YEARS	3 to 4 YEARS	5 to 6 YEARS	7 to 10 YEARS	11 to 14 YEARS
Miliary and meningeal tuberculosis	13	19	28	10	8	2
Chronic and general or pulmonary tuberculosis	7	6	6	1	4	6
Total fatal cases	20	25	34	11	12	8

In Chapter V, page 124, I have offered an explanation for the fact that tuberculosis in childhood may involve organs other than those of the lung as readily as the lung itself, and, in case the lung is involved, the infection may be in other portions than the apex, and will not discuss it here.

What Predisposes a Child to Infection.—The most striking fact which is forced upon us when we consider the amount of tuberculosis present in childhood is that the opportunity for infection is almost universal. One person in every nine or ten dies of tuberculosis and there are about four persons ill of it for everyone who dies. Every person who dies of it is ill and expectorating bacilli on an average of three or four years before death. Among the poor where families are large and people are crowded together and obliged to live under unsanitary conditions, there is probably ten times as much tuberculosis as there is among the well to do; and the poor are the people who are most careless of their expectoration and consequently most dangerous to their fellows. Under these circumstances we are confronted by a situation which offers an almost unlimited opportunity for infection. Contamination through milk; through articles of food; merchandise, through the habits of children crawling on the floor and putting their hands from the floor to their mouths, likewise the habit of children putting things in their mouths and trading toys which have been used by others; and the possibility of infection through flies, are all means by which the bacillus is brought into the body of the child (see Chapter II).

The fact that there is a lack of specific resistance in the small child is also extremely important. Aside from this we have the peculiar structures of the children which are apparently less resistant to infection than those of the adult. Behring has shown that the mucous membrane of the intestine in a small child offers little or no protection against infection. Cornet[4] states that in making several hundred animal experiments he has come to the conclusion that the mucous membranes are more easily penetrated in young animals than in adult animals and he thinks that the reason the infection is carried to the lymph glands in the young is because of the wider lymph spaces as compared with those of the adult. The absence of specific cellular defense in early years is probably more of a factor than the ease with which mucous membranes are penetrated. It is probable that the lymphatic system affords the greatest natural protection of the child against bacteria, and that the child's defense depends almost wholly upon it until such a time as a specific resistance has been developed through contact with specific microorganisms.

Frequency of Tuberculosis in Children.—When the importance of the various tuberculin tests, particularly the cutaneous, intradermal and needle track reactions came to be understood, their ease of application gave us a new method of studying the incidence of tuberculosis in childhood and we were greatly astonished to find how common tuberculous infection really is.

Postmortem examinations in children have for a long time shown tuberculosis to be very prevalent, but they have not been made with sufficient care to show how high this percentage really is. Those, however, which have been made since the universality of the disease has been revealed by the tuberculin tests have come nearer to approaching the pathological statistics.

The tuberculin test has been administered to many groups of children by different observers in all sections of the world, all of whom have come to about the same conclusion. The following tables will graphically show how common infection is, according to various age periods. Table V is from Hamburger[5]

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[4] Die Tuberkulose, 1st ed., Alfred Holden, Wien, 1899.
[5] Kindertuberkulose, Leipzig and Wien, 1910.

TABLE V.

Percentage of Children Reacting to Tuberculin According to Age Period. Test Repeated When Negative (Vienna).

46	children in the	2nd	year of whom	4 or 9	per cent reacted
56	" " "	3rd	" " "	11 " 20	" " "
75	" " "	4th	" " "	24 " 32	" " "
50	" " "	5th	" " "	26 " 52	" " "
63	" " "	6th	" " "	32 " 51	" " "
46	" " "	7th	" " "	28 " 61	" " "
30	" " "	8th	" " "	22 " 73	" " "
35	" " "	9th	" " "	25 " 71	" " "
26	" " "	10th	" " "	22 " 85	" " "
29	" " "	11th	" " "	27 " 93	" " "
19	" " "	12th	" " "	18 " 95	" " "
17	" " "	13th	" " "	16 " 94	" " "
17	" " "	14th	" " "	16 " 94	" " "
509				271	

TABLE VI.

Percentage of Children Showing Tuberculous Infection on Postmortem Examination

AGE	NUMBER OF THOSE EXAMINED	FREE FROM TUBERCULOSIS		INFECTED WITH TUBERCULOSIS		DIED OF TUBERCULOSIS	LATENT TUBERCULOSIS	LATENT TUBERCLE BACILLI
			%		%			
1st yr. 1st quarter..	82	76			6	4		2
1st yr. 2d quarter..	55	46			9	4		5
1st yr. 3d quarter..	36	20			16	10	1	5
1st yr. 4th quarter..	28—	19—	80	9—	20	6—	2—	1—
	201	161		40		24	3	13
2 years..........	65	48	74	17	26	14	1	2
3 "	26	18	69	8	31	4	1	3
4 "	18	12	67	6	33	5	1	..
5 "	16	7	44	9	56	8	..	1
6 "	12	2	20	10	80	8	1	1
7 "	13	7	54	6	46	3	..	2
8 "	20	7	35	13	65	6	6	1
9 "	9	3	33	6	67	3	2	1
10 "	11	3	27	8	73	4	4	..
11 "	14	1	7	13	93	7	4	2
12 "	13	4	31	9	69	6	3	..
13 "	13	4	31	9	69	4	5	..
14 "	13	1	8	12	92	4	8	..
15 "	40	8	20	32	80	19	13	..
	484	286	59	198	41	119	52	27
		484				198		

and is based upon the positive reaction to the tuberculin tests. A failure to react to one test was not taken as negative, but the same test was repeated or another employed, before an opinion was given.

Table VI, shown on page 101, based on postmortem examinations is from Harbitz.[6]

Tables VII and VIII from Fishberg[7] show the ages of the children and the number and proportion giving positive or negative reactions. These represent children in the dispensary who were given only a single test. The proportion of positive reactions is much smaller than that of Hamburger, shown in Table V, a fact which probably is due in part to a failure to repeat the test or apply a new test in case of a failure to react; but which further indicates that infection may not be quite as general in the children of our cities as it is in Vienna.

TABLE VII.

PERCENTAGE OF CHILDREN REACTING TO TUBERCULIN TEST, ADMINISTERED ONCE (NEW YORK)

AGE	NUMBER	POSITIVE		NEGATIVE	
		Number	Per Cent	Number	Per Cent
Under 6 months..	22	1	4.54	21	95.46
6 to 12 months..	34	5	14.71	29	85.29
2 years.........	39	13	33.33	26	66.67
3 "	36	14	38.89	22	61.11
4 "	44	19	43.18	25	56.82
5 "	51	24	47.06	27	52.94
6 "	55	29	52.73	26	47.27
7 "	45	27	60.00	18	40.00
8 "	45	28	62.22	17	37.78
19 "	40	27	67.50	13	32.50
10 "	43	30	69.77	13	30.23
11 "	35	22	62.86	13	37.14
12 "	44	29	65.91	15	34.09
13 "	35	27	77.14	8	22.86
14 "	20	15	75.00	5	25.00
Total..........	588	310	52.72	278	47.28

Tables V and VI from Hamburger and Harbitz make an extremely interesting comparison, the one showing clinical, the other pathological tuberculosis, in European cities.

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[6] Untersuchungen über die Tuberkulose im Kindesalter. 1. Die Haufigkeit. 2. Die Formen der Tuberkulose 3. Angeborene Tuberkulose (Vom pathologische-anatomischen Institut zu Kristiania) Norsk Magazin for laegevidenshapen, No. 1, p. 1-36, 1913.

[7] The Cutaneous Tuberculin Test in Children, Archives of Pediatrics, January, 1915.

TABLE VIII.

PERCENTAGE GIVING POSITIVE REACTIONS

AGE	CHILDREN OF TUBERCULOUS PARENTS		CHILDREN OF NON-TUBERCULOUS PARENTS	
	Number of Cases	Per Cent	Number of Cases	Per Cent
Under 1 year........	33	15.15	56	10.07
1 to 2 years.........	49	55.10	39	33.33
3 to 4 "	90	68.88	80	41.25
5 to 6 "	95	65.26	106	50.00
7 to 10 "	244	71.31	173	64.74
11 to 14 "	181	74.58	134	69.40
14 years............	37	83.79	20	75.00
Total.............	729		608	

While these statistics show the incidence of tuberculosis among the children of the poor who visit the hospitals in crowded cities such as Vienna, Christiana and New York, they do not tell us the conditions in the usual walks of life. I do not know of any considerable group of children belonging to the families of the middle class and the well-to-do which have been examined carefully for tuberculosis. We would not expect the infection, however, to be as general as it is among the poor. Nevertheless, we are probably conservative in estimating that 75 per cent of all children have an infection before they reach the period of adolescence; in fact, it seems almost a miracle that any should escape. My own experience would show easily 90 per cent; but the children that I have examined have been largely those who have associated with tuberculosis in the family. The most interesting and astounding statistics which have come to my notice are those of Jacob, cited on page 79, showing the prevalence of infection in country districts, even where the usually accepted dangers of infection do not exist.

Herbert G. Lampson[8] made a study of infection in a number of families represented in both clinics and private practice, which is worthy of notice. While the inference that so large a percentage of infection has resulted from the investigated open cases may not be warranted, yet this careful study is of great importance in that it shows the prevalence of infection. I quote the summary and conclusion, which is as follows:

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[8] A Study on the Spread of Tuberculosis in Families. Studies in Public Health, University of Minnesota, No. 1, 1913.

"In thirty-three families classed as containing open cases of pulmonary tuberculosis, 173 individuals were examined. Of those examined, 124 individuals showed evidence of tuberculous infection, 41 showed no evidence of tuberculous infection, and 8 individuals were doubtful or suspicious. These suspicious cases were those which showed more or less signs of tuberculous infection but gave a negative von Pirquet test; or not showing physical signs, gave an atypical reaction to the tests employed. All those classed as infected with tuberculosis gave a typical reaction to the tuberculin tests. Of the eight suspicious cases, one has since been declared tuberculous at the University Dispensary and one has had a pulmonary hemorrhage; both were adults and neither is included in the list of tuberculous infections.

"Among the 124 showing evidence of tuberculous infection are the 23 living center cases. Deducting the 23 center cases, we have 101 individuals presumably infected from 33 open center cases, or 3 and 2/33 for each case. Excluding the center cases, 67 per cent of the individuals exposed showed evidence of infection with the tubercle bacillus.

"In four families classed as containing latent center cases, 22 individuals were examined. Of those examined 8 showed evidence of tuberculous infection and 3 were suspicious. Deducting the 4 center cases, we have a spread of infection in 22 per cent of individuals exposed.

"In three families classed as containing healed center cases, 12 individuals were examined. Of the 12 examined, 6 showed evidence of tuberculous infection. Deducting the three center cases, we have a spread of infection in 33 per cent of the individuals exposed.

"In ten families classed as containing non-tuberculous center cases, 56 individuals were examined. Of the 56 examined, one individual showed evidence of tuberculous infection and two were suspicious, an infection of 1.7 per cent of all individuals in the household.

"In five families classed as controls and containing no reported or suspected cases of tuberculosis, 24 individuals were examined. Of those examined, one showed evidence of tuberculous infection, 4.1 per cent of all individuals in the household.

"Dividing all families examined into two classes, tuberculous and non-tuberculous, there were forty tuberculous families, and fifteen non-tuberculous families. In the forty tuberculous families 207 individuals were examined, of whom 138 individuals showed evidence of tuberculous infection. In the fifteen non-tuberculous families 80 individuals were examined, of whom two showed evidence of tuberculous infection and two were suspicious. That is, 66⅔ per cent of individuals examined in tuberculous families showed evidence of tuberculous infection, and 2½ per cent of the individuals examined in non-tuberculous families showed evidence of infection with tuberculosis.

"Among the forty tuberculous families there are ten families containing 54 individuals, of whom every member was examined, and in which every member showed evidence of tuberculous infection.

"Three families containing 12 members, of whom 7 were examined, showed evidence of tuberculous infection in all those examined.

"In the latest group there was only one family of seven individuals, of whom four were examined in whom no evidence of tuberculous infection could be found outside of the center case. Another latent case in a family of four showed only one individual infected, namely, the wife, and this probably a coincident infection as she had been otherwise exposed. No tubercle bacilli have ever been found in the sputum of this center case. In another latent case there were five children and the wife besides the center case, and only one child showed evidence of infection.

"In another latent case where both husband and wife had been reported open cases there was but one child out of five who showed evidence of infection. One of the healed cases showed no spread of infection in a family of four. Another, where the mother is a healed case, shows two out of four children with evidence of tuberculous infection. In this case the history would indicate that the lesion had healed before the birth of the two immune children. In another case, where the mother was diagnosed as an open case, her health greatly improved before the birth of her second child, the oldest child shows evidence of tuberculous infection and the two succeeding children do not.

"The analysis of the non-tuberculous case is simple. The one case of tuberculous infection found in the ten non-tuberculous families, which had been reported to the visiting nurses as tuberculous or suspected families, was the wife of a man who had been reported tuberculous, and had been supervised for a number of years. He had at one time lost weight and at different times had hemorrhages, but no record of tubercle bacilli having been present in his sputum could be found. He showed no signs of a tuberculous lesion, and did not react to the tuberculin tests. The wife had not been a suspected case and showed no signs on physical examination, but reacted to the tuberculin tests. The family of seven children, five of them under six years of age, were without any indication of tuberculous infection. One other case with evidence of tuberculous infection occurred in one of my control families where no tuberculosis was suspected. The woman who showed evidence of tuberculous infection gives a history of having been in delicate health for two years during childhood. She has five healthy non-tuberculous children. . . . . .

"I conclude from the above studies, first, that the spread of tuberculous infection in families where open cases of tuberculosis exist is greater than it is usually understood to be. Sixty-seven per cent of the individuals of these families, excluding the center cases, show evidence of tuberculous infection. In no case where there has been definitely proven exposure of a family to an open case of tuberculosis, no matter what precautions have been taken, have I failed to find a spread of infection. In at least ten cases investigated, the infection has spread to the limit of available material. Every member of these ten families showed evidence of tuberculous infection.

"Second, that in families where no cases of tuberculosis have been found, no matter what the home life or living conditions were, the number of individuals showing evidence of tuberculous infection was small, namely, 2½ per cent.

"Third, that in families where cases of latent tuberculosis exist, the spread of infection is not as great as in families where open cases of tuberculosis are found, 22 per cent against 67 per cent.

"Fourth, that in families where healed cases of tuberculosis are present, the spread of infection is less than in families where open cases exist, 33 per cent against 67 per cent.

"Fifth, that in families where no tuberculosis is found, the number of individuals showing evidences of infection is very small (2½ per cent) in comparison with the families in which open, latent, or healed tuberculosis exists."

Fate of Early Lesions.—The focus of disease which is established when infection occurs, the same as all metastatic foci from this original focus, may take one of three courses: It may go on to the rapid formation of active tuberculosis; it may heal at once, or later; or it may become quiescent for a time to break out later in active tuberculosis. As stated previously the most common course of massive infection during the earliest years of life is to go on to a rapidly fatal issue, the child yielding within two or three months to an active tuberculous process. In case the meninges are involved, the fatal termination comes within a much shorter period. In cases which heal, a certain amount of immunity is established which protects the individual for a longer or shorter time, the length of which probably varies greatly and cannot be determined by any method that we know. In those cases where the disease does not go on to a complete healing but assumes a condition of partial quiescence and then breaks down later with renewed activity, there are factors both within and without the individual which account for this exacerbation. On the part of the individual we assume that for some reason or other the specific defense against the tubercle bacillus is broken down. We know this occurs during the presence of acute infectious diseases, particularly measles and whooping cough, but also during scarlet fever and diphtheria and after pneumonia. In these cases, however, there are probably other elements which enter. I do not doubt that the activity of the glandular system, particularly that of the bronchial glands in those diseases where the pulmonary tissues are involved, has considerable to do with the lighting up of tuberculous foci, which may be in the peribronchial glands. The increased activity of the glands during these infections was pointed out by Weigart many years ago.

In our studies of immunity we must not forget other factors which lower the defense of the individual and which are more or less beyond his control, particularly in the child, such as the surroundings in which the individual lives; the food that he eats; exposure to all kinds of unfavorable conditions, climatic and other; various other diseases which come and from which he recovers more or less slowly; also the physical and mental strain to which the individual is subjected. All things else being equal, the better the condition which surrounds the individual who harbors a latent tuberculous focus, the less the danger of the disease becoming active. It may be said that this is just another way of stating that every now and then the patient's specific immunity is probably broken down.

We cannot help believing that the protective properties of the tissues vary greatly at different times, under different physical and chemical influences, and that this is probably a very important factor in the action of the bacilli which may be lodged within the tissues. We speak of an increased virulence of bacilli. It is not only possible but probable, that after living in the tissues for a long time, bacilli which have been of low virulence may change and assume a virulent type the same as virulent bacilli may become non-virulent. Whether this is due entirely to themselves or whether it is due to conditions on the part of the individual would be a very interesting thing to know; but, at the present time we have no clue by which we may arrive at this information.

The Effect of the Tuberculous Infection Upon the Child.—Let us consider the effects of the tuberculous infection upon the child, even in case the lesion may not be manifest. We must recall that from unhealed lesions toxins are given off intermittently. These toxins not only affect the adjacent structures but their influence may be widespread. They seem to have a special predilection for the nervous system, and through it injure the body cells. A child who suffers from a lesion which is subject to frequent states of activity, nearly always suffers from general malnutrition, a failure to grow and develop, and an unstable nervous system.

Children suffering from these early infections are not always of the so-called scrofulous type. Scrofula is a term which has been used to describe an ill-defined condition among children whose appearance gives an expression of lack of endurance and who suffer from general inflammations of both the skin and mucous membranes. This was formerly thought to be a forerunner of tuberculosis, but its exact relationship to tuberculosis is not known. Some believe that scrofula is always a result of tuberculous infection. Others believe that there is a non-tuberculous scrofula as well as that due to infection. Scrofula is a condition which particularly affects children of the poor, so it looks as though it might be a condition due to either their mode of life or some infection or toxemia of early years. Ponfick[9] and Schlueter[10] in discussing this condition consider it due to anomalies in the structure of the child, particularly that of the lymphatic system. They say that the individual lymph vessels are narrow, while the lymphatic spaces as a whole are large; and through the influence of certain toxins a condition is brought about where an overactivity of the lymphatic system is produced. There is a lymph stasis and a swelling of the part affected. In this condition the vessel walls are more easily penetrated by both toxins and bacteria than they are in normal children, consequently these children are more prone to disturbances on the part of the lymphatic system and are also more prone to general systemic disturbances. Injury to the skin or mucous membrane is followed by a gathering of an extra number of leucocytes and an unusual amount of pus formation.

We often find these children stunted in growth. The infected child, whether he belongs to the so-called scrofulous class or not, does not develop properly if the disease shows perceptible activity. He is not able to endure. He cannot carry on his school work like other children, neither can he endure play as he should. This is often seen among children of tuberculous parents where we know opportunity for infection has occurred; and, if we wait, as was so commonly the custom in years gone by, the child will often go on to a fatal tuberculosis. As adolescence is reached these symptoms are more or less exaggerated. The young boy or girl is not able to endure mental or physical strain. He may be very bright in his studies but he falls back on account of lack of physical and nervous endurance. After this period has been reached the harm has largely been done and it is very difficult to remedy it. A great percentage of these children will go on, if left alone, and develop active clinical tuberculosis. Others will remain free from clinical tuberculosis but be handicapped all through life because of the presence of this infection, though unrecognized. In others the infection finally goes on to active clinical tuberculosis.

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[9] Über die Beziehungen der Skrofulose zur Tuberkulose, Verhandlungen der Gesellschaft für Kinderheilkunde, Aachen 1900, p. 88.

[10] Die Anlage zur Tuberkulose, Wien, 1905.

The Importance of Recognizing Latent or Partially Latent Lesions in Early Life.—Early lymphatic tuberculosis does not receive the attention that it should. Its recognition is in reality the key to the tuberculous problem, so far as both prevention and cure are concerned, and therefore of prime importance.

The important studies of von Pirquet, Hamburger, Wolff-Eisner, Gangenhofer and others have not only furnished clinical confirmation of the pathological fact that nearly every man has a tuberculous infection during life; but, having shown us when this infection occurs, they have shifted the scene of action from adult life to that of childhood. We formerly thought the longer the man lived the greater his chances of becoming infected, but we now know that few that have attained their fifteenth year have escaped infection. Therefore, if we would prevent infection or would cure the infection when it has taken place, we should direct our attention to childhood.

This early infection produces no symptoms that we have as yet learned to recognize until it takes upon itself a fair degree of activity. It is an infection of the lymphatic glands, the chief danger of which lies in the fact that it carries with it the ever present possibility of assuming a state of activity and causing destructive changes in the glands affected, and of extending to new tissues. Even though the glands themselves may not be enlarged sufficiently to be detected by physical examination or the x-ray, they may still be a danger to the patient. This form of tuberculosis assumes its main importance in childhood. Its recognition depends upon the tuberculin test. In the absence of symptoms, should a positive tuberculin test show in a child in whom careful examination reveals no lesion anywhere, then we are justified in assuming that the infection is, as yet, probably quiescent and confined to the lymphatic glands. On the other hand, should there be symptoms or signs such as accompany active tuberculosis, then the condition should be classed as active glandular tuberculosis, a condition which will be described later. The differentiation between these early infections and the next change in the glands which is marked by a multiplication of bacilli and an increased activity in the focus of infection, is of utmost importance to the child and its future health.

While many of these early lymphatic infections do not advance beyond this stage yet a sufficient number of them progress to justify careful consideration. Postmortems on adults show that there are lesions in the apices of lungs in a very large per cent of cases. We cannot ignore the great importance of the fact that Hart shows 64.3 per cent in four hundred autopsies and Naegeli 71.43 per cent in his series. Most of these are unsuspected during life; yet there is no doubt that, did we recognize them and ascribe to them their full power for harm, we would find that in a great many instances the productive capacity of the individual had been lessened, even though no manifest lesions were present. Probably from one-third to one-half of them would at some time show symptoms of clinical tuberculosis and probably not far from one-fifth of the number would die of tuberculosis. Therefore, the recognition of tuberculosis at any stage after the bacilli have gained entrance to the organism furnishes information to the attending physician which is of value to the patient.

Universal testing of children with tuberculin for the presence of tuberculous infection is a measure which commends itself strongly. All who react are known to be infected. This positive reaction, however, should not cause undue concern if the child remains healthy; but, should suspicious symptoms arise, or, should the child fail to grow and develop properly, then further examination should be made; and, even in the absence of positive findings beyond that of a tuberculin reaction, the child should be given the benefit of the doubt.

Children who are suffering from partially quiescent lesions such as I have mentioned above, yield readily to proper care and treatment. They are greatly benefited by a carefully regulated life; fresh air, water, light and sun baths, exercise, and clothing adapted to their needs. Life in the country and in the sunlight is especially valuable for them. It is also gratifying to see how they can be improved by treatment with tuberculin. A prolonged course of tuberculin given children of this type during the age period from five to ten or twelve years, will often be a determining factor in their development. Even after the lung is involved they yield quite readily to proper treatment. The child has the advantage over the adult in that it is in the period of growth and development. This is the period when the body cells should be at their best because they have not yet been injured by excesses of various kinds and by various poisons which have been either taken into the body or elaborated within the body itself; consequently, this is a very advantageous time for the treatment of tuberculosis. If more attention were given the disease at this time there would be a marked diminution in active tuberculosis during adult life; and probably no other factor would have a greater bearing upon the final solution of the tuberculosis problem.

Tuberculosis of the Lymphatic Glands.—The study of tuberculosis of the lymphatic glands is of great importance because of its bearing upon the question of early infection and the gradual development of cellular immunity. Glandular tuberculosis is pre-eminently an infection which takes place prior to the establishment of cellular immunity. Non-glandular infection, on the other hand, is pre-eminently an infection in which the bacilli are implanted in the tissues after a cellular defense has been established, their retention in the tissues depending upon the reaction which occurs between the specifically sensitized cells and the tubercle bacillus.

The behavior of the glands of the mesentery toward the tubercle bacillus is the same as that of the glands which drain any other tissues which come in intimate contact with the bacillus, such as the cervical, peribronchial, and peritracheal, as described elsewhere in these pages.

Tuberculosis is primarily a lymphatic disease. We mean by this that when tubercle bacilli enter an organism which has not previously come in contact with the same; or, which has not attained a high degree of specific cellular defense, the bacilli pass through the mucous membrane readily and, unless destroyed by the lymph elements, are taken to the glands where they form a nidus and set up foci of infection. It will be noticed that, while we do not commonly find glandular tuberculosis in adults, yet the preponderance of infection in childhood is glandular and the greatest amount is found in the first five years of child life, the period in the child's life before the establishment of cellular defense. This is illustrated by the following table showing the percentages of glandular tuberculosis by age periods of cases reported by Woodhead and Carr:

165 INSTANCES OF TUBERCULOSIS OF THE GLANDS REPORTED BY WOODHEAD AND CARR.

PERCENTAGE OF CHILDREN SHOWING TUBERCULOSIS OF THE GLANDS ACCORDING TO AGE PERIODS.

	Age Period	Number of Cases	Percentage of Cases
SIMMS WOODHEAD—100 Cases	1st year	4	4
	1 to 2½ years	33	33
	3 to 5 years	29	29
		—	—
		66	66
	6 to 7 years	12	12
	8 to 10 years	13	13
	11 to 15 years	9	9
CARR—65 Cases.	1 to 5 years	37	57
	Over 5 years	28	43

It is extremely interesting to see that 66 per cent of Woodhead's and 57 per cent of Carr's cases of glandular tuberculosis occurred before the end of the fifth year. Carr also reports 16 or 57 per cent of involvement during the first year in another series of 28 cases.

In order to understand the relationship between the instances of tuberculosis and these glandular infections, these statistics should be compared with the table taken from Hamburger. It will be noticed in this table that about 50 per cent of children are infected by the time they have reached the fifth year of age. After infection has occurred, the importance of glandular enlargement decreases. From this we can see that tuberculosis of the glands gradually assumes less and less importance as infection with resultant cellular defense becomes more general until finally, in adults, glandular tuberculosis is comparatively infrequent and of little concern. At this time of life, in the majority of individuals, the association with tubercle bacilli has been sufficiently intimate to produce infection and sensitize the body cells. When bacilli, either escaping from some focus already present, into adjacent lymph spaces, or passing through the walls of blood vessels, or coming from the outside, become implanted in the tissues, a reaction at once takes place between them and the local cells because of the specific cellular defense which has been developed; consequently, the bacilli are held in check. They do not pass on readily in the tissues nor are they readily carried to the regional glands. They remain *in situ* and fight for their existence at the point of implantation, as shown in Koch's experiments on previously infected guinea pigs, which I have so often quoted.

The Diagnosis of Active Glandular Tuberculosis.—The diagnosis of this form of tuberculosis is of greatest importance in childhood and of particular importance to those who are treating the children of the poor. Any of the glands of the body may be affected, but the cervical and bronchial show infections most frequently, the mesenteric less often.

Tuberculosis of the cervical glands may exist for years. The glands may be the size of peas or as large as walnuts. When the process is active they are often swollen and painful. It is sometimes difficult, even impossible, to make a positive diagnosis, although painful swollen glands massed together, accompanied by symptoms of toxemia, are exceedingly suspicious. If a subcutaneous tuberculin test is given, evidence may be obtained through a focal reaction, by an increase of pain and swelling, but it must not always be expected.

The diagnosis of active tuberculosis of the bronchial glands is not an easy matter. The glands are deep seated and the symptoms produced by them are not definite. The child may show the same signs as those suffering from tuberculosis elsewhere, malaise, lack of endurance, and failure to develop properly. He is apt to have some cough and be especially prone to bronchitis. The temperature curve may show an elevation. In case the glands are very large the cough may assume a brassy, paroxysmal type and pressure symptoms may appear. But this is more apt to occur in adults. Dullness may be found over the 3rd, 4th and 5th dorsal vertebræ and the x-ray may show shadows.

An elevation of the temperature curve in an underdeveloped child, a cough, with more or less tendency to persistent and repeated bronchitis, a thickening at the hilus, as shown by the x-ray, and a positive tuberculin test, are strong evidence of an active tuberculous process in the bronchial glands. A positive prompt reaction to tuberculin is of aid only in showing tuberculosis to be present. It does not indicate where; but, with the other conditions positive, it would point to the bronchial glands as being the probable seat of infection.

One of the best clinical methods of determining tuberculosis of the bronchial glands in children is spinal auscultation of the voice, as suggested by d'Espine.[11] This method is as follows: The patient is required to pronounce some such numbers as "ninety-nine," or "one, two, three," as distinctly as possible, while the examiner listens with the ear or a stethoscope with small chest piece over the cervical and upper dorsal vertebræ. Over the cervical vertebræ the voice is heard distinctly and has clear tracheal characteristics. In normal children this quality of the voice ceases immediately at the seventh cervical vertebra where the lung tissue begins. When swelling of the bronchial glands exists, the tracheal character of the tone persists below this point and may be heard as low as the fifth dorsal. This is the portion of the chest occupied by the bronchial glands and the bronchial character of the sound is transmitted by the swollen glands which surround the trachea and the bronchi, sometimes filling the mediastinal space to the spinal column, producing a good medium for the transmission of the sound. Auscultation by the ear gives better bronchial sound than the stethoscope, but the latter enables one to differentiate more accurately, and likewise causes an accentuation of the vocal fremitus. When auscultation of the spoken voice is uncertain, it is well to have the child whisper "ninety-nine" and listen carefully and systematically from above downwards over the upper thoracic spines in the same manner as mentioned above.

Palpation should not be omitted in the diagnosis of the mediastinal glands. If the examiner, starting at the first dorsal spine with one finger on one side of the spinous process, and another on the other, will carefully palpate the total density under the fingers he will note in the presence of enlarged tracheo-bronchial glands that there is a distinct increase in resistance when that portion of the spinal column is palpated which is on a level with the enlarged glands. I find palpation even more reliable than percussion in many instances because it does not cause confusion by throwing the entire thorax in vibration. Fig. 16 shows the method of palpating for increased density in the mediastinum.

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[11] Early Diagnosis of Bronchial Adenopathy in the Child, British Medical Journal, 1910, iii, p. 1136.

In examining for enlarged mediastinal glands one must bear in mind that their position varies with age. The bifurcation of the trachea is on a level with the third dorsal vertebra or the disc between the third and fourth dorsal at birth, and gradually descends until it reaches the seventh dorsal in the old man of seventy.

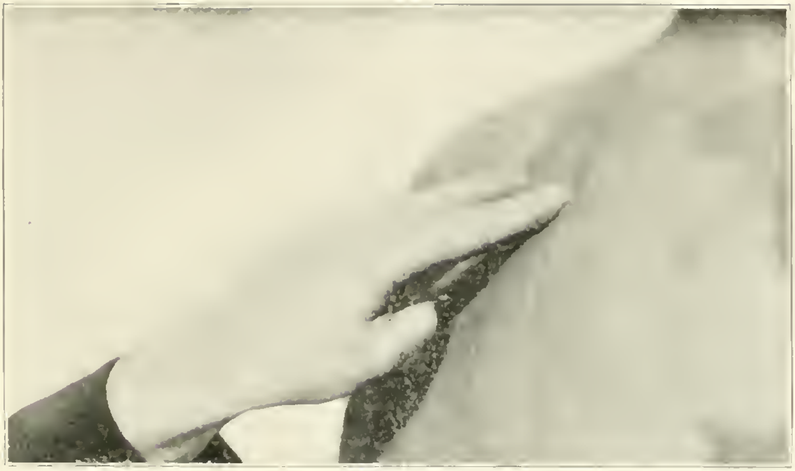

Fig. 16.—Illustrating the method of palpating for increased density in the mediastinum. Increased density is revealed to the palpating finger as readily as it is to the finger on percussion. It shows as a feeling of increased resistance.

CHAPTER V.

FACTORS WHICH PREDISPOSE TO TUBERCULOSIS. WHY THE APEX IS INVOLVED. A CRITICAL STUDY OF FREUND'S THEORY OF THE OSSIFICATION OF THE FIRST COSTO-STERNAL ARTICULATION AND SHORTENING OF THE FIRST COSTAL RING, AS PREDISPOSING FACTORS IN APICAL TUBERCULOSIS.

Disposition and Predisposition.—No disease has been the subject of more speculation and theorizing than tuberculosis. It offers many riddles for solution in every one of its phases. The relationship of human and bovine infection still presents problems which are not understood; the paths of infection; the primary focus; the primary metastasis and methods of spreading; the virulence of the process; the difference in metastases in different tissues; the predilection of metastatic tuberculosis in the adult for the lung and the apex of the lung; the question of immunity; natural and specific resistance; are only a few of the anatomic-pathologic riddles, the solution of which will aid greatly our understanding of this many sided disease.

Speculation as to the specific cause of tuberculosis was set at rest for all time by the work of Koch showing that it is due to a micro-organism. However, this has not settled the question in the minds of all as to whether the tubercle bacillus is the only cause; or whether there are other special predisposing factors on the part of the individual, which are partly responsible for the development of the disease. In order to determine any specific factor which predisposes to infection we must search for something which is common to nearly every member of the race because infection occurs in nearly all human beings sometime during their lives; but this is not the principal question that we are endeavoring to solve in our search for factors predisposing to this disease. We are trying to determine whether or not there is any special condition, whether it be in the physical or chemical constitution or the reactivity of the cell; or whether it be in some special predisposition of certain organs; and whether this is anatomical or whether it is pathological. These problems, it must be understood, from the very nature of the case, are extremely difficult to solve.

One thing is patent to all who are studying this question: that is the fact that the subject of the predisposition of certain individuals and of certain organs to infection seems to receive nearly as much attention today as it did prior to the tubercle bacillus era. Whether this is simply due to the firm impression of old ideas, the impossibility of getting away from tradition; or, whether it is a real problem begging for solution, cannot be definitely settled; but, until settled, it rightfully demands its share of discussion.

Phthisical habitus is practically as old as medicine. Scrofula is almost of equal age. The small heart and narrowing of the arteries has been under discussion for three-quarters of a century. The interest in mechanical predisposition as suggested by Freund in 1858 and 1859, and emphasized later by Hart, and as suggested by Schmorl, Birch-Hirschfeld, and Rothschild is of much later date; while the ideas which particularly have to do with the bio-chemical side of the question are of comparatively recent date.

While the study of disposition and predisposition is extremely interesting and is not without value, yet we must not lose sight of the fact that the mass and virulence of the tubercle bacilli in the inoculation, are factors of tremendous importance in determining not only infection but the course of the disease thereafter. This I have discussed in Chapter III.

Pulmonary Focus.—Metastatic Tuberculosis.—The tubercle bacillus has a tendency, when first taken into the body, to settle in the lymph glands. If it enters through the tonsil or mucous membranes of the mouth it passes at once to the lymph glands that drain these regions. If it passes directly into the lung, along with the inspired air or indirectly through the intestines and thoracic duct, it may form a primary nidus in the lung, but the preponderance of the infection takes place in the peribronchial lymph glands which drain the particular area of lung tissue involved. If it passes through the intestinal wall without producing a distinct local disturbance, it may pass on into and produce infection of the corresponding lymph glands or be taken up by the lacteals, carried through the thoracic duct and poured into the circulating blood to be strained out in the lung or in some other portion of the body, there to produce a local lesion. The character of this local lesion will depend on whether or not the individual has been previously infected. If a primary inoculation, the infection will have a tendency to escape the focus and go on to the neighboring lymph glands; but, if it is a reinoculation, the sensitized cells will attempt to localize the infection in the tissues where first implanted.

Chronic tuberculosis, particularly tuberculosis of adult life, is probably, in the great majority of instances, due to metastases from some previous focus. These metastases occur because, for some reason, tubercle bacilli which are shut up in the glands of the body, become active, multiply, break through the tissues which surround them, gain access to the lymph or blood stream, usually the latter, and find their way to other parts to be strained out wherever the capillaries are sufficiently small to detain them, or wherever the blood and lymph flow is sufficiently retarded to permit them to remain *in situ* long enough to produce infection.

I have little doubt that the greatest factor in the prevention of this secondary spread from the primary lymphatic focus is the patient's immunity, the sensitization of the cells,—the specific cellular defense which has been built up by the presence of the tuberculous focus in these glands. In consequence of this, when bacilli gain entrance to the blood or lymph stream and are carried to new tissues; or, it may be, to distant organs, they meet cells which are sensitized toward them, which have developed specific defensive powers, and stimulate these cells so that they prevent a new infection from occurring. This is probably an important factor in making late childhood comparatively free from metastatic infection in any part of the body and the greatest factor in making metastatic tuberculosis in the adult a chronic disease.

The reason tuberculosis in infants is more often fatal than in adults, and tuberculosis in sucklings more fatal than in older children (as shown by Tables I and II, Chapter IV) is because the child has not had time to successfully fight inoculations of tubercle bacilli and develop a high degree of specific immunity thereby. Specific cell sensitization, the specific body resistance, has not yet been built up, consequently the young child is unable to withstand doses of tubercle bacilli of any great size. Doses of tubercle bacilli which produce rapidly progressive tuberculosis in the infant might gain entrance to the blood or lymph channels of older children or adults and produce no symptoms whatever. So, we might reason, that inasmuch as the specific defense against tuberculosis is a matter of cellular activity, some change in the chemical reactivity of the cell which might be present at times and interfere with cellular activity might prove to be a predisposing factor. But of its existence we are not sure.

We recognize the fact that tubercle bacilli often escape from glandular foci and produce metastases. The figures of Hart and Naegeli show that 63.4 and 71.3 per cent respectively, of adult bodies examined, harbor infections which have been carried to the lungs, usually the apex; but it is a much smaller percentage of these, only about one-third, that show a progressive tuberculosis. It is possible that the progressive cases are due to some lack of cell resistance or some special predisposing mechanical factors; but we cannot get wholly away from the fact that the mass of infection and the virulence of the bacilli are important determining factors in the future progress of the metastatic focus. In our future study of the chemistry of the blood and lymph and the chemistry of the cells of the body and the various problems of cellular reactivity, we will in all probabilities obtain something of value in the solution of these problems.

Commonly Recognized Factors Predisposing to the Formation of Pulmonary Metastases.—The more we study tuberculosis the more we are led to believe that whether any special predisposition towards the disease exists in certain individuals or not, nevertheless any individual may become infected and develop clinical tuberculosis, providing he receives an inoculation of a sufficiently large number of tubercle bacilli.

There are probably many factors which might lower an individual's resisting power and predispose for a time, not only to tuberculosis, but to any other disease. Living in bad surroundings, improper nourishment, overwork and excesses at one time might prove to be the determining factors in an active clinical tuberculosis; when, in other instances, conditions being different, the relationship of the tubercle bacillus to the individual not being the same, they would fail to precipitate the disease. Chronic illness, particularly one which has reduced the patient's strength, might be a predisposing factor not only for infection but for a metastasis to occur and for clinical tuberculosis to show itself. As a rule, however, these predisposing factors must not be looked upon, as far as we know, as being specific. They are only a part of the great number of factors which might appear to favor the development of the disease. Several of them may appear in the same individual at the same time.

The question of racial predisposition to tuberculosis is sometimes mentioned, but there are so many factors which enter into the discussion of this question that it is not fair to say that because a large percentage of a certain race has tuberculosis that it is especially predisposed. It is necessary to look into the life habits of that race, to see how its members live, to see what factors there are which have a tendency to lower their vitality and make them prone to tuberculosis, before such a broad statement can be made. This is true of the negro in America. The conditions under which he works and lives have a tendency to make him more prone to infection. On the other hand we must not forget that in his case there is lacking that strong racial immunity which we find in other peoples whose ancestors have had tuberculosis for centuries. He is one who has more recently come from primitive conditions where tuberculosis did not exist.

The fact that tuberculosis is not common among the members of the Jewish race, excepting among the poorer element is most likely, in part, a racial immunity; but, also in part, due to their habits of life.

If some valid reason can be given why, in adults, the lungs, and particularly the apices of the lungs are nearly always the seat of the infection by the tubercle bacillus, it will help in solving many of the other problems of infection. No matter how the tubercle bacillus gains access to the body, whether through the upper or lower air passages, or through the tonsils or mucous membranes of the mouth, or through the lower intestinal tract, when the time comes for clinical tuberculosis to manifest itself the infection is nearly always in the lung, and not only in the lung but in the apex of the lung; while most extrapulmonary lesions in the adult are metastatic complications from a pulmonary focus.

It is generally assumed that the pulmonary tissue is particularly adapted to the implantation and growth of the tubercle bacillus, the same as the tonsil is adapted to diphtheria, Pyre's patches to the typhoid bacillus, and the skin to the infectious agent of smallpox. Whether this assumption will hold or not will have to be determined. Already a very important fact shakes one's faith in the assumption,—the fact that the primary infection in childhood, if we accept the general belief of pathologists; or, the primary metastasis, if we accept the views of Ghon, is found in the lymphatic glands and not in the lungs. If we believe with Ghon that the primary focus is in the lung and that the glands are secondarily involved, we are still forced to recognize that the lymphatic glands are, because of the numbers of bacilli, or their own function prone to tuberculous infection; for the primary pulmonary focus, as a rule, is small, with little tendency to infect adjacent structures, while the infection after reaching the glands finds soil suitable for multiplication and growth sufficient to cause a considerable inflammation and enlargement of the structures. The parallel between this and the inoculation experiments in healthy animals as first reported by Koch is evident. It might also be suggested that, owing to the relatively large lymph spaces of the child[1] bacilli pass out of the primary focus with such dispatch that they are not given time to become implanted, but are carried to and find lodgment in the glands. That this may be another reason why the glands are so prominent in infection in early life, is further suggested by the fact that the inhalation of dust in childhood shows marked deposits in the subpleural lymph spaces and bronchial glands, but little or none in the lungs, as shown by Shingu[2] while the same experiment in adults shows the lung tissue, particularly the apical tissue, filled with pigment. It would seem that primarily in the child there is a possibility that the lymphatic structures are better adapted to the bacillus than the pulmonary tissue. If this is a fact we must then look for causes other than selection because of suitability of tissue, to explain why pulmonary tuberculosis so greatly predominates over all other forms of disease in the adult.

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[1] Cornet: Die Tuberkulose, 1st ed., Alfred Holden, Wien, 1899, p. 288.
[2] Staubinhalation bei Kindern, Virchow's Archives, Bd. 200.

Let us conceive of tuberculosis as a disease with a primary focus in the lymphatic glands usually in the peribronchial glands, from which metastases are now and then taking place. This conception is the same as that of infection in the tonsil or gums causing metastases, as described by Rosenow, or the metastases in cancer. From the primary focus, bacilli gain access to the blood stream or the lymphatics, and are carried to other parts of the body, the lung, meninges, kidney, liver, bones, joints, in fact, any part or organ of the body. Two factors favor implantation: 1, the bacilli escaping into the vessels are young and often mass together in large clumps; and 2, the reaction between the bacilli and sensitized cells.

That the blood and not the air passages is the chief factor in carrying tubercle bacilli to the apex of the lung is evident from the comparison with air-borne diseases as discussed on page 67. Air-borne diseases affect the base and central portion of the lung, that portion which receives the inspired air most directly and in which the movement of the air is greatest, while the blood-borne diseases affect that portion where the movement is least, giving opportunity for implantation to occur.

Whether tubercle bacilli undergo transmutation while in the body, and this makes those of certain characteristics or certain degrees of virulence, attack one tissue, and others attack another, as is true of the streptococcus (Rosenow), must be left for future study; so must the question of whether or not bacilli of the bovine type are prone to infect certain structures and those of the human type others; and, whether or not, bovine bacilli, by long residence in the human body, change into the human type. There are certain anatomical changes, however, which deserve attention for their influence on the localization of tuberculous infection, even though the different tissues offer varying degrees of susceptibility to infection, and even if it should be found that bacilli undergo transmutation.

Bacilli are capable of penetrating the walls of the blood vessels. The finding of bacilli in the blood of tuberculous patients offers proof of this. No matter whether this takes place through normal vessel walls or only through those which are involved in the inflammation of the tubercle, foci in which bacilli are multiplying are sources of dissemination and danger to the organism. Old foci are now believed to be the most frequent sources of adult tuberculosis (Römer). An old focus in a lymph gland or other tissue may at any time take upon itself a state of active necrosis and set up irritation in the walls of adjoining vessels, permitting bacilli, often in large clumps, to pass through into the lumen and be carried on with the blood stream until destroyed or implanted in some other portion of the body. Aufrecht has described this as taking place and studied sections showing the bacilli passing through the vessel wall.

Once in the vessel, bacilli follow the course of the blood through the heart, lungs and systemic vessels. They may find lodgment in any part of the body; but in fact, in adults rarely find lodgment anywhere except in the lungs until after the lungs are once infected. To account for implantation occurring in other tissue I consider the capillaries of the body, both those of the pulmonary and systemic circulation, as a system of fine screens. In some places injuries occur to these screens, the mesh becoming smaller; in other places it is not improbable that the mesh may be made smaller by pressure. If smaller, for any cause, the mesh offers a barrier to the passage of material of whatever kind that attempts to pass through. The rapidity of the circulation is another factor in passing the screen, the slower the circulation the greater the danger of particles becoming entangled in the mesh.

Localization in the Child and the Adult Differs.—Two patent facts stand out as regards pulmonary tuberculosis in childhood as compared with pulmonary tuberculosis of adult life; first, it is relatively infrequent, and, second, the localization does not show a selective preference for the apex of the lung. In other words, the bacilli are nearly as apt during childhood to be screened out in one portion of the body, with certain limitations of tissues and organs, as in another; and in the lower lobes of the lung as in the upper; as will be seen by the following table from Ghon, showing the localization of primary pulmonary foci.

Ghon's Anaylsis of 170 Cases of Primary Tuberculous Foci of the Lung Shows the Following Distribution:

No. of Cases	Number of cases according to number of pulmonary foci	Distribution of pulmonary foci: Right upper lobe	Right middle lobe	Right lower lobe	Left upper lobe	Left lower lobe	No. of foci	Percentage relationship to entire number of foci
170	142 cases with 1 focus......	44	11	32	33	22	142	71.00%
	15 cases with 2 foci........	10	3	2	12	3	30	15.00%
	5 cases with 3 foci........	3	.....	1	5	6	15	7.50%
	2 cases with 4 foci........	......	.	4		4	8	4.00%
	1 case with 5 foci........	......				5	5	2.50%
	5 cases in which foci cannot be determined ..	?	?	?	?	?	?	?
Number of pulmonary foci divided according to lobes........		57	14	39	50	40	200	
Percentage relationship to entire number of foci...............		28.50%	7.00%	19.50%	25.00%	20.00%		

The study of this question leads us into the realms of both speculation and anatomical facts. After infection has once occurred the child seems to be relatively resistant to the disease, as suggested by the fact that the disease does not regularly spread and form new metastases during this age period. While it is difficult to offer a satisfactory explanation for this, I would suggest that the following are factors. It is probably partly because of the specific immunity which the child has but recently developed; partly because of certain biochemical properties on the part of the tissues of the child which make it more resistant; and partly because the bacilli are not screened out of the blood stream when they gain access to it, but are kept in the circulating fluid until they are destroyed. At any rate, some factors are operating in childhood which do not seem to operate so successfully in later life, which have a tendency to prevent the disease from spreading to any portion of the body, particularly the lungs, even although the infective bacilli are present.

At least a partial answer to the queries why portions of the body other than the lung, in children, are as apt to be involved as the lungs themselves, and why one portion of the lung seems as prone to involvement as another, while the apex of the lung seems especially predisposed in the adult, is suggested by the following anatomical considerations.

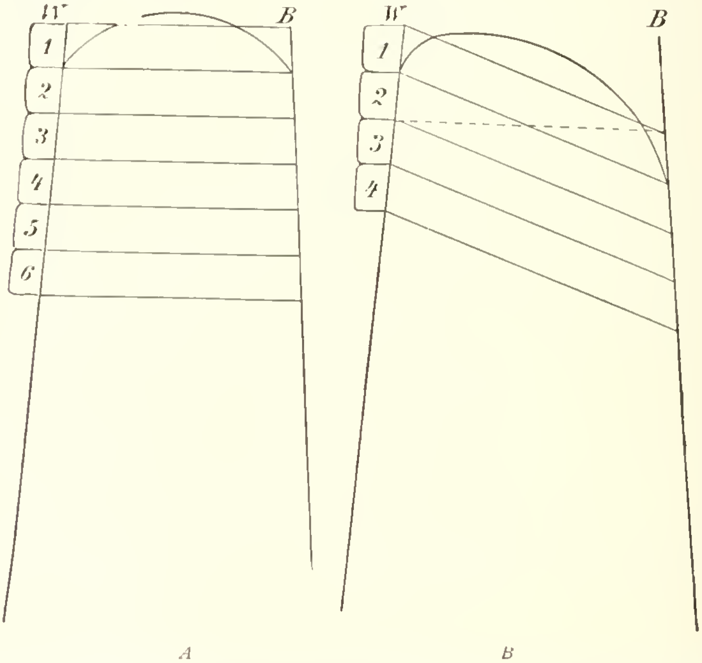

Fig. 17.—Illustrating schematically the difference in the elevation of the anterior portion of the ribs of children and adults. *A*, at birth, first rib on level with first dorsal vertebra; *B*, in adult, first rib on level with third dorsal vertebra.

At birth the anteroposterior diameter of the upper portion of the chest is relatively great, the chest is rotund. The top of the sternum is on a level with the first dorsal (or even the seventh cervical) vertebra. Under natural conditions, the anterior portion of the chest begins to sink during the first year of life, and, by the fifth or sixth year, the top of the sternum is on a level with the disc between the second and third thoracic vertebræ (Piersol) as shown in Fig. 17. Coincident with this descent of the anterior end of the ribs and sternum, the upper portion of the chest flattens. Under pathological conditions this may occur earlier. I have seen a marked pathological flattening of the upper part of the chest, usually unilateral, in children from two to six or eight years of age, where tuberculous infection of the apex existed. This natural flattening which takes place as the child grows older results from the dropping of the anterior portion of the chest wall, as a result of assuming the erect position. Coincident with the descent of the anterior portion of the ribs and sternum, a marked change in the shape of the chest occurs and a change in the respiratory motion of the various portions of the lungs takes place. The round chest of the infant gradually gives way to the wide chest of the adult and the superior portion of the chest becomes relatively flatter than the lower portion. In the child the anteroposterior diameter of the chest below the second costal cartilage is to the transverse diameter as 2 is to 3 at birth, and as 1 is to 2 at three years of age, while it is as 1 to 2½ or 1 to 3 in adult life (Piersol). These relationships at the second costal cartilage, as mentioned by Piersol, may be shown in a way to be appreciated by the following quadrangles (Fig. 18, *A*, *B* and *C*).

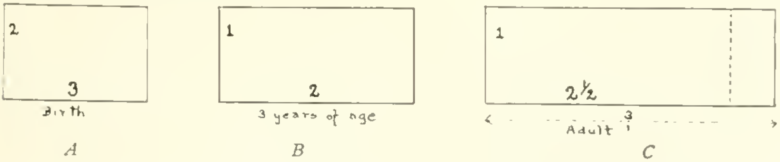

Fig. 18.—Illustrating the relationship between the anteroposterior and transverse diameters of the chest below the second costal cartilage. *A*, at birth; *B*, at three years of age; *C*, during adult life.

The change in the growth of the lung in the adult as compared with the infant is also important. According to Aeby,[3] there are two periods during which very rapid growth of the lung occurs,—the latter half of the first year and the period of puberty. At the end of the first year the lung is four times as large as it is at birth; at eight years, eight times as large; at puberty ten times as large and immediately following puberty, twenty times as large. As the lung increases in size the bony framework above expands the least of any part of the thorax and this emphasizes the compression of the pulmonary parenchyma at the apex, and this, in turn, favors infection at this period of life.

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[3] Der Bronchialbaum der Säugetiere und des Menschen, Leipzig, Wilhelm Englemann, 1880.

The breathing of the new-born child is of necessity abdominal. Thoracic breathing is impossible because of the already high position of the sternum and ribs. Inspiratory effort cannot raise them higher. Bartenstein and Tada[4] say that thoracic breathing is dependent upon and due to the child's assuming the upright position, standing and walking bringing it about.

Gregor, quoted by Bartenstein and Tada, calls attention to the difference in the respiratory compensation in the new-born and the adult. The former can easily double the frequency of respiration as demanded, but cannot increase the depth to any extent, while the adult increases slightly in frequency but more in depth. This is significant in its effect on the mechanics of the thoracic cage. In the former, the movement is great, affecting the entire lung; in the latter the deeper inspiration affects the base and lower portions of the lung to a much greater extent than the upper portion; because at the base the full effect of the contraction of the diaphragm is exerted, as well as that of elevating the larger and more movable ribs.

Mehnert[5] also contributed an important study to this question of developmental and acquired change in the thorax. He says:

"In the primitive bony thorax of the 17 mm. embryo (Fig. 19*A*) the sixth rib lies almost straight and forms an angle of about 90 degrees with the long axis of the body.

"In the child of 4 years (Fig. 19*B*) the inclination of the axis of the same rib has already reached 8°. Inasmuch as this change takes place about equally from year to year during this period, it shows an average change of 2° a year.

"The sixth rib of the 36 year old man (Fig. 19*C*) has already reached an inclination of 26° or an increase of 18° over the 4

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[4] Beiträge zur Lungenpathologie der Saülinge. Franz Deuticke, Wien and Leipzig, 1907.

[5] E. Mehnert: Über topographische Altersveränderungen des Atmungsapparates und ihre mechanischen Verknüpfungen an der Leiche und am Lebenden, Gustav Fischer, Jena, 1901.

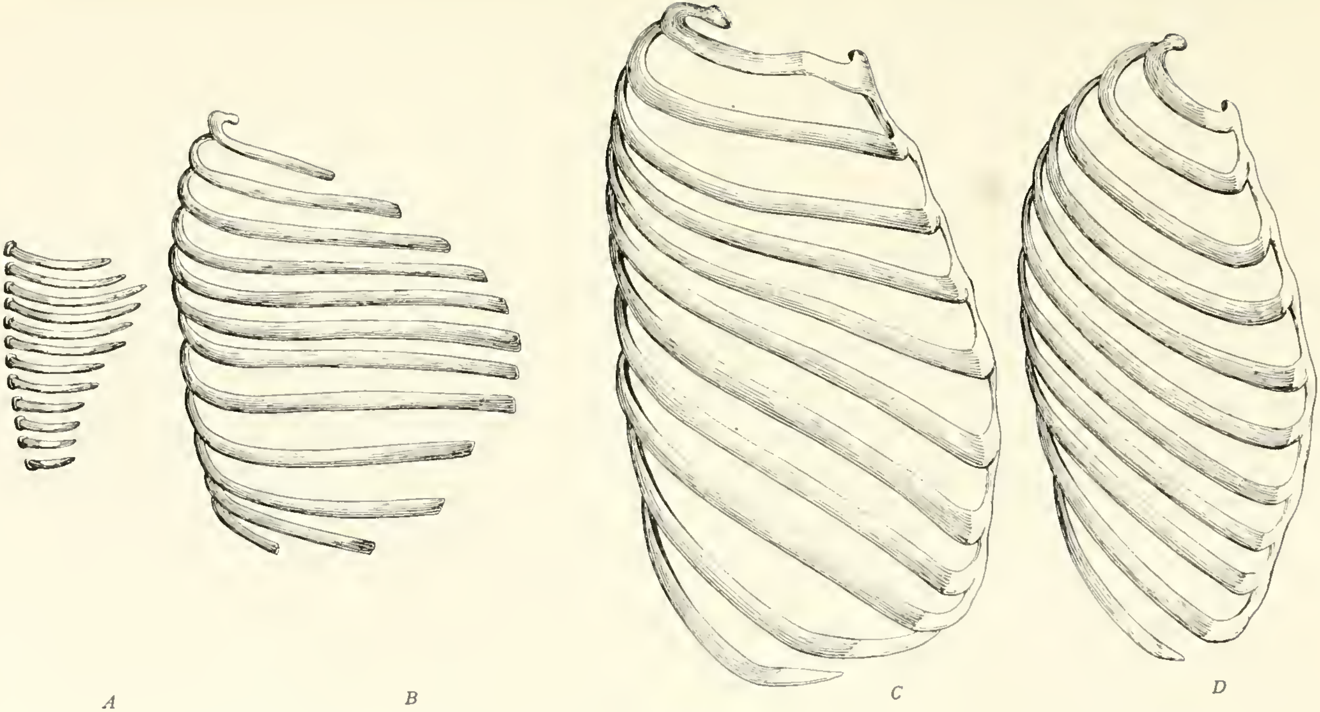

Fig. 19.—Illustrating the inclination of the ribs at various age periods, showing the manner in which the front of the chest drops with increasing age. *A*, in 17 mm. embryo; *B*, in child four years old; *C*, in adult 36 years old; and *D*, in adult 72 years old. (Mehnert.)

year old child, which has taken place during a period of 32 years. The yearly increase in inclination during this period is about ½°.

"The difference in the angle of inclination of the 36 year old man and the 72 year old man (Fig. 19*D*) is 9° which takes place during 36 years, giving a change of ¼° per year.

"These individuals belonging to four arbitrarily chosen age periods show that the inclination of the rib continues during the entire period of life, but that it does not make the same progress at all periods, but decreases with years. In the first years of postfetal life the inclination increases 2° yearly. In the next thirty years the progress of this inclination is four times less. Still less is the progress of the rib inclination in advanced years. It is less than half as great as it is in the previous period and only one-eighth as great as in the first years of life. There can be no doubt that the inclination of the ribs is the direct result of the pulling which the weight of the thoracic organs, the liver, and the filled stomach exert upon the diaphragm and bony thorax."

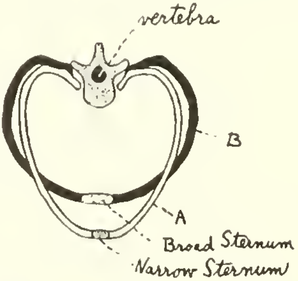

Fig. 20.—Illustrating the relative increased anteroposterior compression of the apex which takes place in man as the result of assuming the erect position, as compared with the four-legged mammals. *A*, four-legged mammal; *B*, man. (Keith.)

It is significant that with the changes in the bony thorax which comes on in man with increasing years, and which results from his assuming the erect posture, comes a change in the localization of the tuberculous infection; also that a similar change is noted in comparing tuberculosis in adults with spontaneous tuberculosis in animals. While the localization in the cow, the other animal prone to tuberculosis, is commonly on the serous membranes, or in the posterior lobe of the lung, or a general tuberculous pneumonia, in man it is almost universally in the apex of the lung. The relative increased anteroposterior compression of the apex which takes place in man as a result of assuming the erect position as compared with the four-legged mammals is shown in Fig. 20.

From the anatomical facts here presented, it can be seen that while the lung is growing and increasing in size; and, as a whole, is becoming functionally more and more active, the growth and activity of the lower portions far outstrip the upper portions; in fact, *while the lung is growing, the upper portions of the bony thorax are flattening, and relatively decreasing in size.*

The effect of this flattening of the upper portion of the chest at a time when the lung as a whole is increasing in size, is an ever increasing compression of the underlying apical tissue and a relative decrease in the total volume of the upper portion of the lungs as compared with the lower portion; also a squeezing together of the lung tissue of this portion, a narrowing of the lymph spaces and air cells and a decreasing of the respiratory movement with its consequent retarding of blood and lymph flow. The relative and actual decrease in the amount of lymph coursing in the apical lesion may also be a factor in reducing the resistance to infection. Whereas the flow of blood and lymph is more or less the same in all portions of the lung in the child, it is embarrassed in the apex of the adult. Consequently, in the child, infection, particularly of hematogenous origin, which is the most common mode of metastatic infection, may occur in any part or organ of the body or any portion of the pulmonary tissue where a capillary is narrowed or retardation of the blood flow is sufficient to permit implantation of bacilli to take place; and is about as likely to occur in one place as another. In the adult, on the other hand, the apex of the lung, which seems mechanically predisposed by its embarrassment and its lessened motion and its retarded lymph and blood flow, is the usual seat of infection. In the early years of life the bony thorax grows faster than the lungs themselves[6] which fact removes the element of compression which is so important in increasing the predisposition of the apex in adults.

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[6] Hermann Pflüger's Archives, vol. xx, 1879.

It is interesting to note that these changes in the contour of the chest take place at the same time that pulmonary tuberculosis begins to assume a relatively greater importance, as compared with the meningeal, peritoneal, and other forms which are more common in early years. Clinical pulmonary tuberculosis does not become an extremely common disease until after the second decade; and this term of years between early childhood when infection is occurring, and adult life when clinical tuberculosis manifests itself in the pulmonary apex, marks a time in the life of the child when it should show a natural resistance and is also the time when the child which has become already infected with the tubercle bacillus, is best protected by the specific immunity which it has developed.

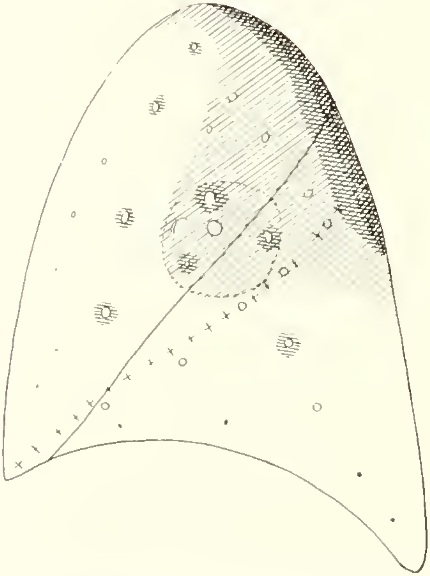

Fig. 21.—Illustrating schematically the movements of the various portions of the lung. The degree of motion is indicated according to the shading—the darker the portion, the less the motion. (Tendeloo.)

Consideration of the predisposition of the apex of the adult lung to tuberculous infection has called forth a great deal of discussion and many theories; but, for the most part, the suggested theories have been based on anomalies rather than on natural anatomic and physiologic considerations.

The theory of Freund that ossification of the first costal cartilage and shortening of the first rib through pressure upon the apex predisposes to tuberculosis, likewise that of Rothschild in assuming that a diminution of the manubrio-sternal angle is a predisposing factor, and the furrow described by Schmorl over the posterior portion of the apex, have called forth a great deal of discussion. Against these theories is the very important physiologic fact that one of the most important inspiratory acts is the contraction of the diaphragm which enlarges the lung not only in the anteroposterior direction, but in the superoinferior di-

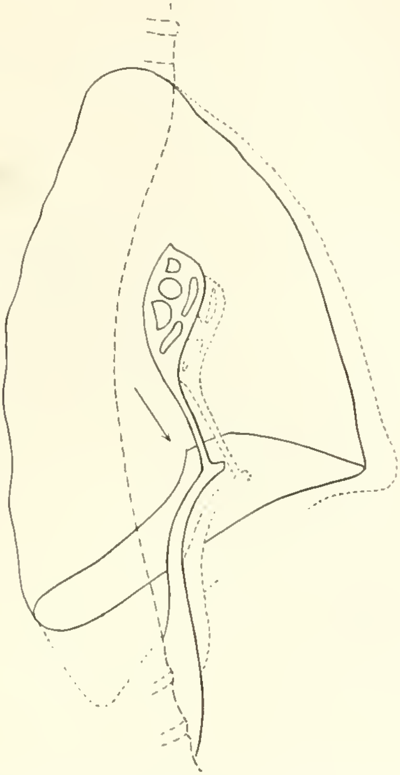

Fig. 22.—Illustrating the mediastinal aspect of the right lung, which shows the degree of respiratory movement at the root. The crus of the diaphragm is indicated and its attachment to the root of the lung through the pericardium shown. The black lines show the lung on expiration, the dotted lines on inspiration. It will be seen that the motion about the hilus is not great, and that at the apex is probably the least of any portion of the lung. (Keith.)

rection as well. Keith[7] called attention to the accordion action of the interlobular sulci of the lung and the general enlargement which takes place with the contraction of the diaphragm.

I regard the factors which make the apex of the lung especially predisposed to tuberculous infection, as more fundamental than the above mentioned theories would indicate.

Apical Compression Following Anatomical Growth Slows Blood and Lymph Current.—A key to the situation lies in the fact that the apex of the child's lung is not predisposed to infection, while the apex of the adult lung is so predisposed. The solution of the problem may be aided by studying the anatomic and physiologic changes in the chest during the period of growth, and by studying the action of the inhaled dust at the various age periods, as previously mentioned.

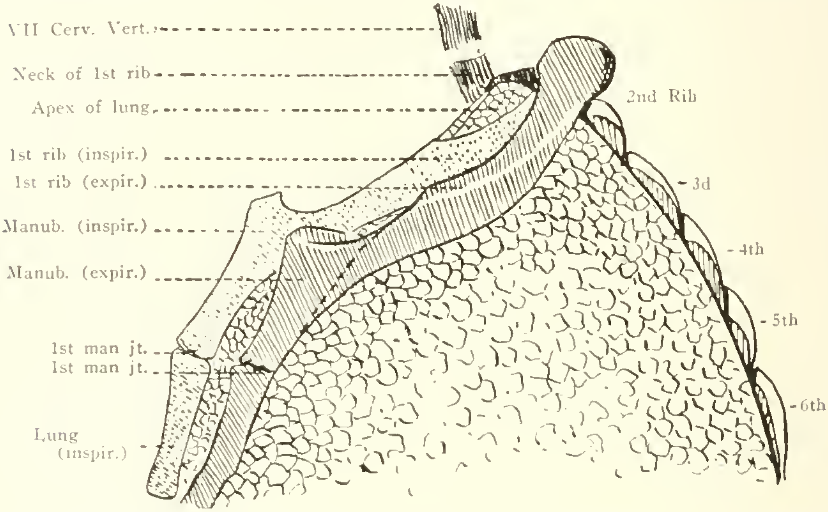

Fig. 23.—Illustrating the action of the first pair of ribs and manubrium in increasing the area of the lung during inspiration. Showing that the posterior aspect of the apex is the portion subjected to least movement. (Keith.)

To repeat the important facts mentioned elsewhere in these pages, if the child inspires air surcharged with dust or soot the particles do not lodge in the pulmonary tissues but pass through to the subpleural lymph spaces, and more particularly to the peribronchial lymph glands. In other words, they follow the course of the lymph in the lymphatic channels. In the adult lung, on the other hand, much dust and soot is deposited in the lung tissue itself, and in the subpleural spaces, while the bronchial glands are less affected. It is further observed, in the adult lung, that dust and soot are deposited particularly in the apex of the lung, while the base is comparatively free. It has been suggested that the reason for the marked deposit of soot in the lung tissue in the adult is because, through protracted exposure, more and more particles have become deposited in the lymph spaces and that these retard the progress of others taken in later. It has also been thought that there is more or less irritation and thickening of the lung tissue caused by the dust particles which prevent their rapid transfer to the lymph glands. The narrowing of the lymph spaces must also be considered. Some have suggested that these deposits in the tissues act as a predisposing cause to the localization of the tubercle bacillus.

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[7] Further Advances in Physiology, Edit. by Leonard Hill, 1909.

Tendeloo particularly, has insisted that the principal factor in predisposing the apex to tuberculosis is the fact that there is a lessened motion of the lung in this area and consequently a retarded lymph and blood flow. This is shown in Figs. 20, 21, 22, and 23. This is a very important consideration in any theory that is offered. Its effect can be seen in the distribution of the deposits of dust and soot in the lung of the child as compared with the lung of the adult, and its influence on the implantation of tubercle bacilli must be equally important.

Critical Examination of Theories of Freund, Schmorl and Rothschild.—I have endeavored to offer an anatomic and physiologic explanation for the fact that the apex of the lung of the adult is predisposed to infection, but I now wish to take up the matter at this point and discuss it in relationship to Freund's theory, which has received so much attention during recent years. With Freund's theory we must also class the theories of Schmorl,[8] Birch-Hirschfeld[9] and Rothschild[10] because they are all closely related.

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[8] Zur Frage der beginnenden Lungentuberkulose, Münchener medicinische Wochenschrift, 1901, No. 50.

[9] Birch-Hirschfeld: Über den Sitz und die Entwicklung der primären Lungentuberkulose, Deutsches Archiv für klinische Medicin, Bd. 64, 1899; Ibid., Des erste Stadium der Lungenschwindsucht, Bericht Tuberkulosekongress, 1899.

[10] Über die physiologische und pathologische Bedeutung des sternalkinkels, XVIII Kongress für inner Medicin, Karlsbad, 1899.

Freund[11] published his first articles with reference to anomalies of the upper aperture of the thorax as factors in the causation of tuberculosis in 1858 and 1859, but his theories were unnoticed for forty years. No interest was taken in them until Birch-Hirschfeld published his paper with reference to the peculiarities of the post apical bronchus and its relationship to tuberculosis and Schmorl brought out the idea of the bronchi being compressed by the furrows, which are produced by the upper ribs encroaching upon the lung.

It was then that Freund[12] himself revived his previously suggested theories and with the aid of his pupils made for them a prominent place in the literature of tuberculosis.

These publications created new interest in the anatomo-pathologic conditions surrounding the apex of the lung which during recent years have assumed great importance in the discussion of the question as to why the apex of the lung is predisposed to tuberculosis.

The discussion of this subject by Hart[13] and by Hart and Harrass,[14] and Baemeister[15] are extremely important and set forth in a clear manner the arguments in favor of these predisposing factors.

As representing Hart's recent discussions I will translate a quotation from his article in Bei-heft No. 11, zur Medizinischen Klinik, 1912, as given by Martius[16] in a recent discussion of this subject.

" 'The upper aperture of the thorax which is formed by the first ribs and vertebra and sternum is heart-shaped in form. On either side of the vertebra are the apices of the lungs which are surrounded by the bony portions of the first ribs. The costal ring departs from the horizontal in such a way that while the apices of the lungs are about on a level with the junction of the first rib with the vertebra, the anterior end of the first rib is 2½ cm. below the apex. In contrast to all of the other true ribs we find that the first costal cartilage has no articulation with the sternum. In its place there is a short broad cartilaginous connection, whose union is made secure by the special firm consistency of the cartilage. On inspiration the cartilage takes upon itself a spiral action of such a nature that the motion produces a strong tension, the release of which permits, or mechanically forces the costal rings to return to the position of expiration. The first costal ring itself is elevated during inspiration, its position approaching the horizontal, but it produces no widening of the upper aperture. On forced inspiration, even, the respiratory movement of the upper aperture is also relatively small in comparison with that of the remaining ribs, which gradually increases toward the base and whose elevation and elastic expiratory motion is favored by their free articulation with the vertebræ, their long elastic cartilages, as well as their articulation with the sternum; yet, in spite of this the function of the first costal ring is of much greater importance in the respiratory movement than that of all the others.

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[11] Beiträge zur Histologie der Rippenknorpel in normalen und pathologischen Zuständen, Breslau, 1858; also Der Zusammenhang gewisser Lungenkrankheiten mit primären Rippenknorpelanomalien, Erlangen, 1859.

[12] Thoraxanomalien als Predisposition zur Lungenphthise und Emphysem, Verhandlungen des Berliner medizinische Gesellschaft, Nov. 27, 1901.

[13] Die mechanische Disposition der Lungenspitzen zur Tuberkulösen Phthise, Stuttgart, 1906.

[14] Der Thorax phthisicus, eine anatomisch-physiologische Studie, Stuttgart, 1908.

[15] Entstehung und Verhuetung der Lungenspitzen Tuberkulose, Deutsche medizinische Wochenschrift, 1911, Nr. 30.

[16] Handbuch der Tuberkulose, Brauer, Schröder und Blumenfeld, vol. i, 1914, p. 409,

" 'The respiratory motion of the lungs is regulated by a law which has been suggested by Tendeloo, that every portion of the lung is dependent functionally upon that portion of the thorax overlying it. According to this, while distension of single portions of the lung is of influence upon the amount of the entire volume of respiratory air, compensatory balances for local loss taking place, one portion of a lung is never able to influence the functional activity of a distant portion in such a way that the movement of the lung tissue in all places is uniformly the same. The alveoli of the apices of the lung are, in a general physiologic sense dilated to a lesser degree during inspiration; and the total pulmonary tissue experiences a weaker expansion than is the case in the remaining portions of the lung. Only by a forced pronounced costal breathing does the lung apex show a strong respiratory motion. By adjusting respiration to the minimum demand of oxygen, as in the usual moderately shallow thoracic breathing, a complete filling of the apices of the lungs seldom takes place; consequently, under normal conditions, the respiratory motion of the upper part of the lung, when compared to the lower portion of the lung, that is, the prevailing relative functional activity, is decidedly small, as has been shown by Riegel.'

"Under normal physiologic conditions, this aeration is sufficient, but, under pathologic conditions, it is different.

" 'The important fact of our new teaching is that in almost every adult person suffering from tuberculosis there is an anatomical departure from the normal in the upper aperture of the thorax. At one time this takes the form of abnormal shortening of the cartilage as described by Freund; again, in an arrested development of malformation of the bony portions of the rib; and, still again, in a combination of both of these conditions. Sometimes the changes are symmetrical and sometimes asymmetrical, but always produce the same effect, differing only in degree; namely, the transformation of the aperture from the oval heart-shaped form into a more long oval form, as is the type in animals and in man during the fetal stage. In the formation of this pathological aperture the ribs do not run in a gentle curve, but more directly forward to the sternum, consequently, the transverse diameter is shortened; but, what is of paramount importance, there is present a greater or lesser contraction of the posterior para-vertebral areas in which the apices of the lungs lie and move.

" 'The upper costal ring which shows these pathological changes can only be considered as a whole and in its relationship to the entire thorax. One must recognize that not only its pathological configuration, but the degree of narrowing of the superior aperture of the thorax is extremely variable, and also that it depends somewhat upon the condition of the neighboring parts (vertebra). He must also see that the anatomical changes of form and changes of position of the aperture and its relationship to the expansion of the apices emphasizes any anatomic functional disturbances that might be present. This disproportion produces a permanent pressure upon the pulmonary tissue of the apex. The consequence of this continuous pressure of the narrow and immobile costal ring, which is elevated only a little or not at all through the respiratory motion, is an injury of the lung tissue which is thus encircled. This makes itself manifest subapically by means of a very marked furrow in which the ribs lie. In the interior of the lung, however, the small bronchi are compressed in such a manner that the apical bronchial systems give one the impression of being stunted in growth. Whoever has seen these two changes which Schmorl and Birch-Hirschfeld first considered worthy of description, must be completely convinced of their dependence upon the changes in the first costal ring. Whoever opposes this has never seen these changes in the pulmonary apices and forgets that in a large material of thousands of autopsies only an accident could bring it before one's eyes, even though he were continually looking for it, because primary tuberculosis quickly destroys the original anatomical relations.

" 'What we must conclude from these positive anatomical facts is plain. The mechanical functional disturbance of the upper aperture of the thorax leads to an interference with the ventilation of the apical tissue, and, secondarily, to an embarrassment of the blood and lymph flow, both of which are, in a large measure, dependent upon the respiratory movement of the pulmonary tissue. The interference with the ventilation of the apices is not only logically explained by the functional dependence of the pulmonary apices upon the respiratory movements of the upper aperture of the thorax, and the specially unfavorable conditions which result during quiet respiration from the peculiar course of the apical bronchi; but can also be directly proved upon the living through shadows indicative of thickening of the tissues shown röntgenologically, which indicates partial or complete atelectasis or a later stage of collapse induration, and also on the cadaver by an increased deposit of carbon in the apical tissues.

" 'Arnold has experimentally shown, and it is likewise a common postmortem experience, that foreign particles are usually lodged in the neighborhood of thickenings in the tissues, also in the tissues which do not take full part in the respiratory movement. I (Hart) have especially called attention to the fact that the initial tuberculous apical lesion does not correspond, as a rule, to the sub-apical pressure furrow and the costal ring, but occurs in the tissues which are partially atelectatic.

" 'From a physical standpoint the likelihood of tubercle bacilli settling in the apical tissues is also increased by the aperture anomalies. The reason that the bacilli are able to exercise their specific injurious influence is because of the increase of the biochemical susceptibility in the tissues, in consequence of the disturbance in the blood and lymph circulation which causes them to lose their natural resistance to the tubercle bacillus in consequence of poor nutrition. That such a thing exists no one can doubt who recognizes that almost every man comes in contact sometime with tubercle bacilli, and that they penetrate into the lung, usually in comparatively small numbers, nevertheless he becomes ill and suffers either from progressive tuberculosis or from some of the less virulent forms of infection. It has also been shown that the farther the upper aperture departs from the normal the quicker the process in the pulmonary apex spreads after infection has occurred, and the more rapid does it spread. On the other hand, the susceptibility of the tubercle virus, the individual disposition, is the same without reference to the way the bacillus enters the body, whether by the air, the blood or lymph passages. The physical opportunity and the biochemical susceptibility is the same. Aside from this the conditions under which the tuberculous process spreads in the inferior portions of the lung and in other organs has nothing to do with the question of disposition.' "

Martius asks the question: how is this theory to be harmonized with the work of Ghon which shows that in children the apex does not show a special disposition to tuberculosis? And answers it by quoting from Hart as follows:

"In children the fixation of the upper thoracic aperture, which is the result of a gradual development, has not yet taken place.

"With the completion of the growth of the body, every functional disproportion between the development of the thoracic aperture and the development of the pulmonary apex becomes exaggerated, and of greater importance the wider the departure from the normal. At this time both the physical conditions and the biological susceptibility favor the implantation and growth of the bacillus no matter when or in what manner it enters. The morbidity from tuberculosis increases with great suddenness (explosionartig) at the end of the second and beginning of the third decade and the disease does not heal but goes on making an almost continuous selection from all of those who are predisposed. Now and then someone will escape infection with the tubercle bacillus and its consequences; some, by possessing relatively favorable conditions in the pulmonary tissue are enabled to develop a resistance to the enemy, which lasts for a long time, probably through life; in all severe cases, however, the battle is decided and the fate of the individual sealed from the beginning."

The weak point in these theories lies in failing to grasp that any compression caused by a primarily shortened rib or narrowed aperture or changed manubrio-sternal angle, is only a part of the general compression which occurs in the upper portion of the thorax in the adult, as compared to the child, thus basing infection of the apex upon anomalies instead of natural anatomic and physiologic factors.

There should be added to this excellent recent description given by Hart, some of the ideas which were originally enunciated by Freund, which are not here mentioned.

In Freund's[17] early description in discussing the ankylosed rib, he particularly calls attention to the altered condition of scaleni muscles in the following language:

"In many patients who are suffering from beginning chronic tuberculosis of the apex of the lungs, one finds pathological changes, especially in the first costal cartilage, this change being an ossification. The bony change does not proceed from an inflammation of the pleura, for it usually begins first on the edges, then on the outer surface of the rib, and at last on the inner surface, and all takes place without the pleura being changed. This also occurs in beginning tuberculosis where the pleura is not yet involved. Under these conditions one always sees strong antagonistic development of the scaleni whose point of insertion on the rib often shows an exceptional size."

My recent clinical observations[18] on the presence of reflexes caused by inflammation within the lungs shows that these muscles are thrown into definite contraction (spasm) reflexly from the disease within the lung, and that what Freund described here was not hypertrophy due to muscles pulling against an ankylosed rib, but a reflex contraction due to the pulmonary inflammation.

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[17] Beiträge zur Histologie der Rippenknorpel in normalen und pathologischen Zuständen, Breslau, 1858, quoted by Hart.

[18] Muscle Spasm and Degeneration in Intrathoracic Inflammations and Light Touch Palpation, C. V. Mosby Co., St. Louis, 1912; and Inspektion, Palpation, Perkussion and Auskultation bei der Frühdiagnose der Lungentuberkulose, Brauer's Beitrage zur Klinik Tuberkulose, vol. xxxiii, part 1, 1915.

Trophic reflexes also arise from the lung; these express themselves visibly as a degeneration of the skin and subcutaneous tissue of the neck and chest above the second rib anteriorly and the spine of the scapula posteriorly; and of the accessory muscles of respiration. I have suggested trophic change also as being responsible for the dry arthritis of the shoulder joint and the change in the upper thoracic aperture as described by Freund. The first and second ribs and their costal cartilages are supplied by cervical nerves, fourth to eighth; the first by the fourth cervical which lies in the most direct path of pulmonary reflexes. It is plain therefore that this trophic reflex, caused by the tuberculous inflammation in the lung might result in early ossification of the first and second cartilages (particularly the first), ankylosis of the chondrosternal articulation, and limited bony growth. It is extremely suggestive in this connection that Hart should say:[19] "Freund attempted to carry this interference with the development of the first costal cartilage back to the time of childhood, even to the fetal epoch, but I, personally, have in no case, either in a child or in a fetus been able to find unquestionable symmetrical shortening of the first costal cartilage as a result of some prenatal interference with nutrition of the bone and cartilage." That tuberculous infection has a decided reflex trophic influence on these structures we know from clinical experience and are led to infer from the analysis of Hart's own statistics; and, that it could cause ankylosis of the first costosternal articulation by reflex trophic action and the diminished motion which follows infection is quite probable.

At this point I desire to quote from papers by Stiller, Schiele, and Révèsz, because they have important bearing upon the relationship of compression of the upper aperture of the thorax to tuberculosis, also upon the relationship between calcification of the first costal cartilage and the tuberculous infection.

Stiller[20] questions the importance of the thorax phthisicus as being a predisposing cause of tuberculosis and also claims that the anomalies of the upper aperture of the thorax are identical with his thorax asthenicus. He does not consider that the early ossification of the first costal cartilage upon which Freund and Hart lay so much weight is more than one factor among many.

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[19] loc. cit.

[20] Der tuberkulöse Thorax phthisicus und die tuberkulose Disposition, Berliner klinische Wochenschrift, 1912, No. 3.

Schiele[21] lays particular stress upon the degree to which the rib departs from the horizontal, claiming that the greater the angle of departure of the upper aperture in asthenic individuals, the greater the interference with the function of the organs within. He looks upon the early ossification of the first costal cartilage as not being a cause but a consequence of apical tuberculosis and considers that it is due to a diminution of the intensity of the respiratory movement. He says that thorax phthisicus is not inherited but developed after birth.

Révèsz[22] reports the examination of 22 individuals with marked thorax asthenicus (Stiller) and found, according to the method of Harris, a shortening of the first costal cartilage in every instance. For controls he examined 11 people with normal chests and did not find a shortening of the first rib in a single instance. In 22 asthenic individuals he found only 4 in whom there was no trace of tuberculosis. In these 4 there was also no ossification in the first costal cartilage in a single instance. In 7 of the remaining 18 who had a tuberculous infection, the apex of the lung was normal, the infection being in the peribronchial glands. In these 7 cases ossification of the first costal cartilage was also wanting. In the remaining 11 cases which showed pulmonary tuberculosis radiologically, calcification of the first costal cartilage was found, therefore Révèsz draws the conclusion that it is not the asthenia but the tuberculosis which produces the calcification in the costal cartilage and that this is not a predisposing cause but a result of tuberculosis. He further states that there was no evidence of ossification in the first costal cartilage in any of the 11 controls, and, further, that ossification of the first costal cartilage has not been found in a single instance in the Rœntgen Institute of the University of Budapest in a young individual who did not also at the same time have pulmonary tuberculosis. On the contrary, not a single instance of progressive tuberculosis has been examined which did not show an ossification of the first costal cartilage.

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[21] Die Neigung der oberen Thoraxapertur, Zeitschrift für klinische Medicin, 1912, vol. lxxvi, parts 5 and 6.

[22] Thoraxphthisicus und Thorax asthenicus, Gyögyászat, 1912, no. 5; Internationales Centralblatt für die gesamte Tuberkulose-Forschung, 1912, VII, Jhg., no. 2, p. 67.

Of the 400 bodies which he examined postmortem with reference to shortening of the first costal cartilage, Hart found in 114 or 28½ per cent that they were shorter than normal. Of the 114 bodies showing changes in the first rib, 104 or 26 per cent of all suffered from tuberculosis of the pulmonary apices and 78 or 19½ per cent of the total number suffered from progressive tuberculosis. He states that this shortening of the rib has a definite effect in producing progressive tuberculosis because 78 or 62.4 per cent of the 125 progressive cases found in the total 400, showed abnormal shortening of the first costal cartilage. How it could do this is difficult to explain. The suggestion of deficient nutrition does not satisfy. This fact is a great deal more suggestive to me of progressive tuberculosis being a cause of the changes in the rib and cartilage, as I shall discuss later. According to the theory which justifies artificial pneumothorax, compression should favor quiescence rather than progressive tuberculosis.

In Hart's 45 cases which were intimately associated with tuberculosis in the family 34 or 75½ per cent showed shortening in one or both cartilages, a larger percentage than in non-tuberculous families. The fact that tuberculosis shows in a larger percentage of those who come from tuberculous families than those who come from non-tuberculous families and that shortening of the cartilage is also more common in these, would indicate that this anomaly seems to be increased with increased danger of infection. The infection also extends to the lungs more often and earlier in children who associate with open tuberculosis in early life because of the greater exposure and more massive infection; therefore, the reflex trophic effect produced on the costal cartilages and the effect of lessened motion would be far more apparent, and we would also expect this anomaly to be more common if it were a result of the disease.

It is well known that tuberculosis of the lungs is not common in childhood and that if it occurs it usually goes rapidly to a fatal termination; nevertheless, the more massive the infection, the greater the danger of pulmonary metastases occurring. This explains the fact that more pulmonary infection is found in the children of families where there has been open tuberculosis than in the families where less danger exists. Mediastinal glands produce a trophic reflex affecting the superficial structures the same as the lungs. With the more massive infection, and the probability of earlier extension to pulmonary tissue and the consequent greater inflammatory reaction, we are led to suspect a more marked trophic reflex in the children of tuberculous families than in those free from such association. The greater percentage of trophic change in the cartilages therefore, is not surprising.

The most suggestive part of Hart's statistics to me, however, is the comparative relationship between the incidence of infection and the anomalies of the first costal ring. He found that 254 or 63.4 per cent of the 400 bodies examined postmortem showed apical infection, but only 114 or 28½ per cent showed pathological changes in the costal cartilage; and that, further, in 78, or 62.4 per cent of the 125 cases of progressive tuberculosis, shortening of the first rib was found. This would indicate that these changes in the costal cartilage are not particularly related to *tuberculous infection, but that they are related to active progressive tuberculosis.* We know that active progressive tuberculosis, as a rule, is a chronic disease and as such would have greater opportunities to produce trophic changes in the costal cartilage than would abortive infections, such as those which are so commonly found in the apices of the lungs, but which never go on to active development. Likewise, active progressive tuberculosis would favor ankylosis of the costo-sternal articulation by the reduced respiratory excursion which accompanies active pulmonary disease.

These anomalies cannot be discussed except by taking into consideration the mechanics of respiration.

Tendeloo[23] in his careful study of the mechanics of respiration has shown us that the air cells in different portions of the lungs are expanded unequally. There is only a slight expansion of those near the hilus, a little greater in those at the apex, and still a little greater in the para-vertebral area of the superior lobe. These three areas of the lung are particularly those of diminished respiratory movement. The air cells of the lower lobe are subject to greater inspiratory effort and consequently are dilated more. Two forces must be considered in respiration. One, the costal rings and the other the diaphragm. The costal rings are so attached to the vertebral column and the sternum that during inspiration not only is the anterior end raised but the lateral portion of the ribs is also elevated (this is especially true of the lower ribs), thus increasing the transverse as well as the anteroposterior diameter of the chest. The axes of these costal rings depart from the horizontal in different degrees. While the first costal ring departs only about 10 degrees from the horizontal, the tenth departs nearly 45 degrees as shown in Fig. 18. Resulting from this, there is a marked unequal movement of the various portions of the thoracic wall above and below, the movement of the superior being slight while that of the inferior is quite marked. When we add to this the contraction of the diaphragm we have another force which enlarges the thoracic cavity in the vertical direction and which exerts its greatest influence on the lower portion of the lung. The change in position of the diaphragm on inspiration and expiration amounts to from one to two inches. Its contraction not only lengthens the thoracic cage but also helps to force out the ribs and increases the transverse as well as the anteroposterior diameter of the lower portion of the chest. Our knowledge of the relative value of diaphragmatic and costal breathing in air capacity is not well established. Tigerstedt[24] says relative to diaphragmatic and costal breathing that we have at present but a single measurement of the absolute value of the diaphragmatic as compared with the costal enlargement of the thorax; namely, out of 490 centimeters of inspired air in man about 320 centimeters develop upon the elevation of the ribs and only 170 centimeters on the descent of the diaphragm.

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[23] Studie Über die Ursachen des Lungenkrankheiten. Wiesbaden, 1901.

While we may roughly say that the expansion of any particular portion of the pulmonary tissue depends upon the motion of the thoracic wall immediately over it, as suggested by the law of Tendeloo, quoted by Hart, yet this is not wholly true, because the action of the diaphragm exerts its influence even to the apex of the lung as shown by Keith.

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[24] Text Book of Physiology. D. Appleton & Co., New York, 1906.

Keith[25] says that these physiological facts (referring to the motion of the pulmonary tissue and the distention of the air cells in the various parts of the lung) have a very important bearing upon the localization of the tubercle bacilli and the predisposition of the lung to infection. Tubercle bacilli find most favorable conditions for implantation in these areas of lessened lung expansion. This fact has been used to support the theory of Freund, but it seems to me that there are many points which show that the cause of the tendency for tubercle bacilli to be implanted in the apex or the posterior aspect of the superior lobe is more fundamental than shortening of the first costal cartilage and its compressing influence upon the pulmonary tissue. If it were due to the compression from the rib we would expect to find the implantation occur not posteriorly where it so often does, but laterally where compression is greatest.

In reality the movement of the first pair of ribs and the manubrium, as Keith shows, expands particularly the anterior or ventrolateral part of the apex of the lungs, particularly that part of the lung in front of the neck of the first and second pair of ribs, where implantation so commonly occurs; so it seems to me that since there are other ways of satisfactorily accounting for the ankylosis of the first costo-sternal articulation and shortening of the first rib and costal cartilage are apt to be results of, and not caused by, tuberculosis, and since the implantation of bacilli does not occur where we would expect it to occur from the nature of the case, if it were due to the action of this first costal ring we are forced to look for some other cause which operates to favor apical infection. This greater cause, I believe, is found in the changes which take place in respiration as the child grows in years and adolescence is approached.

The following facts seem of prime importance to this discussion:

1. Apical infection is favored as the child grows older by the dropping of the anterior ends of the ribs which compress the apex and slow the blood and lymph movement.

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[25] Further Advances in Physiology, Hill, London, 1909.
[26] Die Anlage zur tuberkulose, Wien, 1905.

2. Bacilli rupturing into the blood stream from active foci are young and often form in large clumps. These are readily detained in the compressed vessels in the apex, which favors implantation.

3. Implantation is further favored by the specific reaction which takes place between bacilli and the antibodies present in the pulmonary tissues.

4. Ankylosis of the first costo-sternal joint and shortening of the first rib may be due to a trophic reflex resulting from tuberculosis in the underlying lung which is in reflex connection with this rib and cartilage through the sympathetics and the third and fourth cervical nerves.

Habitus Phthisicus.—This subject has received attention from many writers, particularly Schlüter.[26] There has been described a certain form of body which has been thought to have a predisposing influence to tuberculosis; but, now that we understand the tuberculous infection and its effect upon the human body better, we know that in many instances *habitus phthisicus* is not a predisposing factor to tuberculosis but a result of tuberculosis. We further know that while this peculiar build furnishes its just proportion of victims to tuberculosis, yet there is no form or shape of body that is not susceptible to tuberculous infection. Moeller[27] says that any practitioner knows that at least three-fourths of his tuberculous patients have a normally built thorax. My own experience would confirm this view. The principal characteristics of *habitus phthisicus* are a long flattened chest with wide intercostal spaces, prominent clavicles, long thin neck, acute costo-sternal angle, winged scapulæ, with a tendency to scoliosis. The individual is usually tall and has weak musculature. To this has been added more recently the anomalies of the upper aperture of the thorax (Freund) as well as that of a floating tenth rib.[28]

The Small Heart.—It is necessary in dealing with the special factors which predispose to tuberculosis to give attention to the fact that for many years the size of the heart has been considered as a causative factor. While I have discussed the cause of this more fully elsewhere in these pages, it is necessary to mention it here in connection with the subject under discussion, but I would refer those interested to the fuller discussion on page 301.

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[27] Lehrbuch der Lungentuberkulose, Wiesbaden. 1910.

[28] Stiller: Die asthenische Konstitutionskrankheit, Stuttgart, 1907.

Brehmer was convinced at the autopsy table that the small heart was present in a large number of tuberculous subjects and was convinced that it was a predisposing factor in the production of the disease. He reasoned that, the heart being small, the lungs were proportionately large for the amount of blood they received, consequently, on account of undernutrition, became diseased. It must be remembered in reading Brehmer's work that his ideas were given out in the prebacillary days. His belief in the existence of the small heart in tuberculosis was based on 12,000 careful personal observations.

It was found that the small heart existed not only in the bodies of those who came to autopsy, as a result of their tuberculosis, but in those who died of accidental death and in whom an early or moderately advanced infection was found. In such cases the small heart was attributed to some defect of development and was naturally considered as having a causative relationship to the disease.

My suggestion for the cause of the small heart is a physiological one.[29] It is based upon the fact that aside from the strength of the heart muscle and the elasticity of the vessel walls, the greatest accessory factor in the circulation of the blood is the suction action caused by the inspiratory act, which accompanies the enlargement of the thoracic cage. With every inspiration the negative pressure in the thorax is increased, the vessels are opened and the blood is drawn from the systemic veins and delivered to the heart. Any condition which interferes with inspiration reduces this suction action and consequently causes less blood to be delivered to the heart. The heart, receiving less blood, contains less blood and delivers less blood at each systole. The result is that it accommodates itself to the condition and becomes smaller.

The same cause operating, causes the relatively small arteries which likewise have been described as being predisposing to tuberculosis.

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[29] The Small Heart in Tuberculosis: A Physiological Explanation, Journal American Medical Association, April 17, 1915.

CHAPTER VI.

THE NERVOUS SYSTEM IN TUBERCULOSIS.

Psychoses.—The nervous system shows very interesting and important changes in tuberculosis. These departures from the normal affect the nervous system at all levels, physico-chemical, sensorimotor, and psychical. The relationship between tuberculosis and insanity has long been recognized. The death rate from tuberculosis among the insane of asylums is double or quadruple that of the general population. This is not so difficult to explain now that we know the frequency of infection in the human race and also know that the advanced lesions of later years may be due to renewed activity in old foci. The lowered resistance of the insane can readily bring about conditions favorable to the multiplication of bacilli; and when activity has once started, the patient offers feeble resistance to the spread of infection.

In some instances, on the other hand, the tuberculous process with its toxins is most likely the active force in the production of insanity. I have seen several cases of insanity which were complications of active pulmonary tuberculosis in which the tuberculosis seemed definitely to be the etiological factor.

Jessen is of the opinion that diseases dependent on degeneration of the nervous system are more common in tuberculous families than in the non-tuberculous.

A very interesting psychical disturbance which has come under my observation on several different occasions is that of double personality. I have seen a number of patients who, a short time prior to death, became extremely confused and thought themselves two different individuals. In one instance the patient kept expectorating carelessly about on the bed clothing and floor, and, when reproved for it, said that he was two individuals and that the other fellow was careless while he used the cup. Such patients sometimes think that one personality is a long way off and the other one present. I have noticed this as a terminal symptom, coming on during the last few weeks of life, except in one instance under my care at the present time. This patient, a young woman, began to suffer from active symptoms accompanied by fever about four months ago. One month later she was having chills with high fever, and suffered greatly from the cold. In describing her conceptions, she says that she herself was always comfortable, but that she could not keep the other personality warm no matter how much cover she used. She also thought of the other person as doing all of the coughing and expectorating, while she did none. This patient is suffering from advanced caseous tuberculosis with fever and a complicating tuberculous enteritis. (She died two months after this was written, six months after first noting the double personality.)

Psychoneuroses.—More frequent than the psychoses are the psychoneuroses and neuroses. Tuberculosis often develops in individuals of the nervous type. Sometimes this nervous condition is inherited. In those of the enteroptotic type (*asthenica universalis congenita*), Stiller claims that the nervous characteristics are present from birth but become manifest more particularly after puberty. In other instances the nervous symptoms gradually appear as a result of toxins which develop in the focus of infection.

In many children tubercle toxins injure the nervous system, destroying the nerve balance; and, through this, interfere with proper physical development. This harmful influence on the nervous system is often carried on for a prolonged period before frank clinical tuberculosis develops. It is often difficult, under such circumstances, to tell whether the nervous condition or the tuberculosis existed first.

The nervous side of tuberculosis has impressed some authors so thoroughly that they look upon it as being a nervous disease. One can readily understand how the toxemia of tuberculosis can produce a vicious circle by its action upon the nervous system and, through it, interfere with the physical development. Realizing that one of the important functions of the nervous system is to preside over the nutrition of the body cells, the effect of a more or less constant toxemia, such as that produced by chronic tuberculosis, is evident. By its action on the neurons there is a constant disturbance in nerve impulses which must be conducted to the tissue cells through the nerve filaments. It is quite possible that this is the true cause of the degenerations which result from toxemia.

While, as yet, we are not wholly able to explain the action of toxemia, yet we are able to say that its force seems to be spent directly upon the central nerve cells and the endocrine system, the effect of which is shown in disturbed function. While our knowledge of the physiology of the nervous system would not permit us to believe that the sympathetic neurons alone are involved, yet there is a peripheral expression through the sympathetic nervous system. We must conceive that the chronic toxemia produces more or less constant stimulation of the nerve cells of the central nervous system. Resulting from this they become more or less irritable, fatigued and later, exhausted. This irritability is shown in dysfunction on the part of the organ receiving its nerve impulse from the cells which are involved. One particular action caused by toxins is its stimulation through the sympathetics of the adrenal gland with an increased production of adrenin. This, again stimulates the sympathetic tissues peripherally and prolongs the action produced by toxemia. While the toxins act upon the central nerve cells, adrenin acts peripherally at the myo-neural junction. With stimulation of the sympathetics, either centrally or peripherally, we have a general inhibition of function throughout the important internal viscera which produces an extremely deleterious effect upon the organism as a whole.

The irritability of the nerve cells shows itself by a lowering of the threshhold of response, so that a stimulation which, under ordinary circumstances, would be withstood by the nerve cell without resultant action, calls forth an action more or less intense.

From this description it can be readily seen that the psychoneuroses of the tuberculous are characterized by lack of endurance and increased irritability.

Nearly every adult who suffers from clinical tuberculosis has a more or less pronounced neurasthenia, which becomes more marked as toxemia is prolonged. These patients suffer from malaise. They are weary even without exertion and tire unduly upon the least effort, whether of a physical or mental nature. Added to this is often a complete change in nature, an irritability, a lack of self-control. A quiet nature may become easily angered and even quarrelsome. Dependence is also a common symptom. Patients who have been self-reliant often lose their confidence in themselves. They like to have things done for them, and may even see their friends make unusual sacrifices without show of appreciation.

The nature of the tuberculous often becomes much like that of the child,—selfish, self-centered, irritable, easily angered and easily pleased.

McCarthy and Carncross[1] present a careful study of the mental attitude of the tuberculous patients treated under their care during the years 1904-5. The mental attitude of these patients might be somewhat different from that of the patients in the well-to-do class, but the analysis is very interesting. Carncross says:

"The question, then, must be not are tuberculous people particularly optimistic or hopeful or cheerful or anything else, but do they undergo a change of mental state? And this I think may be answered reservedly in the affirmative—so reservedly, indeed, that one must say that generally the change is due to chronic illness rather than to tuberculosis specifically. Having claimed this freedom from specific influence, one must promptly whirl about and assert that in a certain number of cases the disease does not affect the brain markedly,—that here are found more or less characteristic mental states which are over the borderland. But of these I shall speak later on.

"Well, then, in the long run, what are the changes, if there are any, in disposition or mental state that the tuberculous individual does undergo? They are what one would expect from the condition of the general nervous system, which is one of irritability—the irritability of weakness. The spinal reflexes are almost universally increased before the very advanced stages have been reached, in a limited number of which, as Doctor McCarthy has shown, they are abolished. And this same irritability is evident in the brain. The vast majority of tuberculous patients will promptly admit that they are irritable since they have been afflicted with the disease. They frequently describe themselves as 'cranky.' With this, in many cases, is a greater tendency to worry, though here, of course, circumstances, which have become so much more unfavorable, play a large part. But the sense of apprehension and the dread of trouble, as well as the susceptibility to annoyance, are increased independently of external conditions. The patient often admits that he is less cheerful and less sociable, and that his outlook tends more toward pessimism. On the other hand, he may simply be more indifferent, or quieter, or have become entirely ambitionless. Any one, two, or all of these changes may be present in the tuberculous individual."

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[1] Second Annual Report of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis, Philadelphia, 1906.

The following tables which are self-explanatory, show the natural disposition of the patient and the attitude after tuberculosis has developed (McCarthy and Carncross). They do not bear out the universality of the condition of *spes phthisica* which seems to be so generally accepted.

DISPOSITION.

Patients with	Cheerful temperament	155	237	Some change in disposition	148	(18 non-tuberculous)	237
	Unhappy temperament	22		No change in disposition	58	(5 non-tuberculous)	
	Intermediate temperament	29		No record of change in disposition	31	(7 non-tuberculous)	
	No record of temperament	31					

PRESENT MENTAL ATTITUDE.

Patients who were	Hopeless regarding disease		166	(16 non-tuberculous)	237
	Not hopeful regarding disease		32	(4 non-tuberculous)	
	Indifferent regarding disease		1		
	No record		38	(10 non-tuberculous)	
Patients who were	Depressed	Most of the time	62	(6 non-tuberculous)	237
		At times	49	(10 non-tuberculous)	
		Seldom	14	(1 non-tuberculous)	
	Not depressed		71	(6 non-tuberculous)	
	No record		41	(7 non-tuberculous)	
Patients who were	Suspicious		10	(2 non-tuberculous)	237
	Not suspicious		184	(20 non-tuberculous)	
	No record		43	(8 non-tuberculous)	

Five of the one hundred and eighty-four recorded as not suspicious were questionable.

The tuberculous patient is generally looked upon as being one of unusual hope. I have never been able to make up my mind that he is far different in this respect from what he was before his illness. One optimistic when well, is apt to be when ill of tuberculosis, and there is surely nothing in this disease that can turn a pessimist into an optimist. If we consider the seriousness of the disease we must marvel at the hopefulness of those who are inclined to be of a hopeful nature; on the other hand the confirmed pessimist is, at least, as hard to cheer and encourage when tuberculous as when well.

There is one factor which acts by inspiring hope all along through the course of the disease. That is that there are spells of illness, the disease becoming more active than usual and being accompanied by temperature, chills, and other symptoms of toxemia, increased cough and expectoration, which are followed again by a remission or cessation after a short time. They see this so often that they expect it, and consequently learn to look forward to the time when they will be better, no matter how ill they are. While this is the attitude of the optimist, the pessimist often greets his physician, who is trying to encourage him, by such statements as: "Yes, but how long will it last?" or "Yes, but I have been this way before and it always gets worse." Compared with the patient with severe acute illnesses, in which toxemia is a factor, or other chronic diseases accompanied by toxemia, I think the tuberculous patient is in about the same frame of mind during his periods of acute activity, and during lesser activity, as one with other chronic toxemia; but, of course he is hopeful and buoyant, if an optimist, when the toxic symptoms have passed off. If a pessimist he is always pessimistic. I have never been able to determine that the tuberculous patient, with severe gastrointestinal complications is any less depressed than the nontuberculous with the same symptoms.

The tuberculous patient is often described as one who is particularly credulous and easily duped. In this way do some account for the fact that he is preyed upon to such an extent by quacks, charlatans, and the unscrupulous. Is he more easily duped than the average human being? Before answering this we must recognize that he is suffering from a serious fatal disease. Our profession has offered him little hope, in fact, has told him time after time, that it can do nothing for him. Naturally, he exercises his right, if not his judgment, and turns to the thing that does offer hope. It is doubtful whether this is an indication of excessive gullibility. Is it not just as apt to be a bowing to the principle of self-preservation, the first law of nature? Are they more gullible than others who follow fake advertisements not only in medicine but in every-day transactions? Are they more easily duped than other intelligent people who blindly follow cults and fakers?

The fact has been cited that they are so easily preyed upon that any new measure for relief is accepted with unusual eagerness and that every suggestion of itself is apt to be noted. This may be explained on the ground that relieving fear and discouragement and substituting hope in their place relieves the patient of nerve irritating influences and improves his general tone, as noted on page 224. This is not peculiar to the tuberculous. The nontuberculous can also be put to sleep at times by a hypodermic of sterile water; so can they be relieved of pain by the same.

The human race, as a whole, is influenced by suggestion, and to this the tuberculous patient is not an exception. One of the greatest factors in handling the tuberculous patient is suggestion. This factor is most important in its influence on the nervous system. The nervous system controls every function of the body, so its effect is far reaching. Positive suggestion is a powerful force and should always be employed. It should be so positive too that it will overcome all negative suggestions that can come from outside sources. It is difficult to measure the influence of suggestion upon the cure of tuberculosis, but it cannot be denied that proper suggestion will relieve sympathetic inhibition, produce a calm state of mind, induce sleep, improve digestion and assimilation, and have a favorable influence upon the heart and circulatory system; but, this it will do for the nontuberculous as well as tuberculous.

There are many circumstances, when numbers of tuberculous patients are under close observation, that make it appear that they lack will power and are extremely easily led. In my experience I would say that three-fourths of the patients who have confidence enough to seek my aid are willing to cooperate to the best of their ability, while the others need considerable attention to hold them to conscientious work. In discussing this in a former paper[2] the writer said: "The patients who enter an institution belong to three classes. The first comprises the great majority of patients and consists of those who want to do what is right, who are conscientious and earnest in their desire to get well. To be sure these, at times, fall from grace; but, most of the time they are working conscientiously for recovery. Then, there is a small class, probably about twenty per cent, who have no well defined principles, but who are willing to be conscientious if they fall in with the conscientious class, and who are just as willing to be insincere if they are led that way. Then there is still a small number, less than five per cent, who are vicious, willful and unfaithful to themselves and all with whom they come in contact. This class, however, can often be controlled by patience and firmness."

I am not sure that a lack of cooperation on the part of the few can be attributed to lack of will power. It is rather thoughtlessness combined with the usual disregard of other people's rights and an unwillingness on their part to submit to authority. It may be exaggerated over what it would be among an equal number of well people, yet of this, I am not sure.

Von Muralt[3] speaks of the suspiciousness of the tuberculous patients and notes how they mistrust particularly those who are trying to do for them. He also speaks of their egoistical nature, their lack of regard for others. He speaks of how at times they will suddenly become dissatisfied with everything that is being done for them; and says that every sanatorium physician has met epidemics of complaints against the administration and dissatisfaction with the methods of treatment which were not only unfounded but amounted to base ingratitude. Such attacks as these are accounted for on the ground of toxic disturbances of the nervous system. There is no doubt of the effect of the toxins upon the nervous system, yet such behavior has nothing to do with tuberculosis itself. True, it may be somewhat accentuated by it; but would not these same tendencies show themselves in any group of individuals who were under similar disappointment and discouragement, and under the same restraint; and would not they manifest themselves in any group of individuals suffering from chronic toxemia from any other cause?

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[2] Some of the Problems of Private Sanatoria for Tuberculosis as Observed During Ten Years' Experience in the Pottenger Sanatorium for Diseases of the Lungs and Throat, Boston Medical and Surgical Journal, vol. clxxi, no. 4, pp. 142-147, July 23, 1914.

[3] Die nervösen und psychischen Stoerungen der Lungentuberkulosen, Medizinische Klinik, 1913, N. 44 and 45.

Insomnia is often a symptom which seems to be exaggerated in tuberculosis. This is very apt to occur in people who are of a nervous temperament. An obstinate insomnia which fails to yield to ordinary and even extraordinary doses of hypnotics, is a very common symptom which comes on a few weeks before and persists until death.

Hezel[4] quotes the following statistics of marked instances of psychoneuroses in tuberculosis. He himself found it in 30 to 40 per cent, Phillipi in 66.1 per cent of all patients of which there were 52 per cent in the first stage; 59 per cent in the second, and 76.6 in the third stage.

The ultimate dependence of these neurotic symptoms upon toxemia may be inferred, however, from the fact that they are often markedly increased during acute activity and disappear with its quiescence. During the time that necrosis and cavity formation are occurring we, at times, find these symptoms very pronounced and see them let up simultaneously with the rupture and expulsion of the contents. These symptoms, although very pronounced when the patient is living under unhygienic conditions, are, at times, relieved at once when the patient is put at rest under a hygienic regime. The fact, however, that many of them can be markedly relieved by suggestion, indicates that toxemia is not the only factor. The effect of depressive emotional states such as pain, discouragement, fear and disappointment must be taken into consideration in this connection. For a more complete discussion of this interesting phase of the subject see Chapter VIII. There seems to be considerable habit in all cases of psychoneuroses; although, to begin with, there is some physical foundation in practically all instances.

The relationship between neurasthenia and tuberculosis is gradually becoming better recognized. The toxins of tuberculosis are especially prone to affect the nervous system. Now that we begin to appreciate the fact that tubercle bacilli taken into the body during childhood may not produce active tuberculosis until later in life, we begin to understand how these infections may be able intermittently to give off enough toxins to impair seriously the nervous system, without producing more active symptoms. Every observing clinician can probably recall cases of so-called neurasthenia which have, upon careful analysis, proved to have a partially quiescent tuberculosis as their causative factor. This can be understood by realizing that the toxins constantly bombard the cells of the central nervous system and irritate them to the point of exhaustion.

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[4] Tuberkulose und Nerven System, Handbuch der Tuberkulose, Brauer, Schröder und Blumenfeld, Bd. iv, 1914.

It is extremely important to bear this in mind, particularly in young girls and boys during the age of adolescence. In normal individuals this should be the period of exceptional development and increased strength and power. We find quite a proportion of young people, who, instead of taking upon themselves this extra growth, seem to be unable to measure up, and seem to gradually lose in strength and power instead. This is the time those suffering from *asthenica universalis congenita* (Stiller) begin to show this stigma most. It is the time when the so-called neurasthenic condition begins. While there are undoubtedly many individuals whose lack of nervous and physical force is a congenital factor, yet there are many others who develop this after birth, showing it particularly after the age of puberty.

I wish to emphasize that the tubercle toxins which are given off from the semi-quiescent foci which are often present in early life and adolescence are responsible for many cases of neurasthenia. This is true to such an extent that tuberculosis should always be considered as one of the etiological factors in neurasthenia coming on at this time of life.

I have seen many young people, particularly girls, who were gradually lapsing into this neurasthenic state, who have been relieved and practically made over by discovering that the tuberculous toxemia was the underlying cause and relieving it by proper treatment.

Pathology of Psychoses and Psychoneuroses.—Dupré,[5] quoted by Hezel, speaks of the pathology of the psychoneuroses and psychoses in tuberculosis. He mentions a patient who came to postmortem who, during the last few months of life, had suffered from light characteristic attacks of depressive mania. He found over the frontal lobe slight thickness of the meninges without adhesions, also a slight hydrocephalus, but no signs of tuberculous infection. Histological examination using Nissl stain showed a simple meningitis, not inflammatory in character; and without diapedesis. In the brain there was a slight proliferation of the epithelium of the vessels, pigmentation around the vessels, and, in the tissues surrounding the capillaries, an occasional columnar cell. Scarcely any increase of the glia cells was observable. In the ganglion cells of the frontal lobe there were marked degenerative changes, also destruction of the white substance. Chains of strepto-bacilli were seen around some of the vessels. In the other portions of the cortex these processes were much less marked. Dupré considers them as purely toxic, neither of a meningeal nor bacillary nature.

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[5] Euphorie délirante des phthisiques, Etude anatomaclinique Revue neurol, 1904, No. 16, Ref. Neurologisches, Centralblatt, 1904, p. 1164.

Levastine,[6] quoted by Hezel, reports that he has found cell degeneration in the cortex of tuberculous patients who had shown evident psychoneurotic disturbances during life; and has noted marked pigmentation of the cortical cells in cachectic patients. These reports are very interesting because they show the extent of the toxic influence of this disease.

Tuberculosis and the Peripheral Nerves.—The influence of tuberculosis upon the peripheral nerves has received some study during recent years, although for the most part it has been neglected. A nerve may either be influenced directly by the tuberculous process in adjacent tissue such as occurs when the vagus nerve is bound down by enlarged mediastinal glands, or the phrenics are involved in apical adhesions or the intercostals are involved in instances of pleural adhesions; or, as pointed out by Gustav Liebermeister[7] the nerves themselves may be influenced through the direct action of bacilli. The toxic influence has been previously mentioned and is undoubtedly of considerable importance. Reflex action upon the peripheral nerves has particularly interested the writer for a number of years. The disturbance in the larynx in early tuberculosis is sometimes thought to be due to the recurrent laryngeal being compressed by enlarged mediastinal glands. I think, however, that this is more apt to be a reflex hoarseness which appears in tuberculosis and presents two different pictures upon physical examination. In one instance there is an inability to adduct. This is due to interference with the action of the recurrent laryngeal. In other instances the cords approximate at the ends but lack tone and consequently do not approximate in the middle. This is due possibly to interference with the action of the superior laryngeal. I am inclined to think that these processes are due to a reflex stimulation from the disease in the lung, the irritation starting in the inflammation in the lung, the afferent impulse traveling to the brain and coming back through the superior and inferior laryngeal nerves. I have gone into the question of reflexes more fully elsewhere and would refer my reader to page 385.

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[6] Recherches histologiques sur l'écorce cérebrale des tuberculeux Revue de Med., No. 3, 1907, Ref. Jahresbericht d. Neurol. u. Psychiatr.

[7] Studien über Komplikationen der Lungentuberkulose und über die Verbreitung der Tuberkelbacillen in den Organen und im Blut der Phthisiken, Virchow's Archiv für pathologische Anatomie und Physiologie und für Klinische Medicin, vol. xix (series 19, vol. vii), part 1-2-3.

In 1912 the writer reported several cases of acute neuritis regional in character which resulted from tuberculosis.[8] Two of these involved the brachial plexus. Since that time I have had seven others under my care, making nine cases of brachial neuritis in all, which were confined to the side of severest involvement and greatest activity in patients suffering from pulmonary tuberculosis. The fact that they were confined to the side of the involvement; or, where both sides were involved, to the side of greatest activity, warrants the belief that they were directly related to the disease. In my papers dealing with muscle spasm as a motor reflex[9] I called attention to the fact that these nerve lesions are not a part of the general nerve disturbance which might be due to toxemia, but have a definite regional character. The fact that this regional relationship can be traced anatomically to an association between the cervical nerves and the sympathetic nerves which supply the lung leads us to the conclusion that they are reflex in nature. Patients having tuberculosis suffer from sensory, motor, trophic and secretory disturbances of reflex character and in some of these instances the

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[8] Chest and Shoulder Pains in Pulmonary Tuberculosis, Transactions of the American Climatological Association, 1912.

[9] Muskelspasmus und Degeneration, ihre Bedeutung für die Diagnose intrathorazischer Entzündung und als Kausalfaktor bei der Produktion von Veränderungen des knochernen Thorax und leichte Tastpalpation, Brauer's Beiträge zur Tuberkulose, Bd. xxii, part 1, 1912; Muscle Spasm and Degeneration in Intrathoracic Inflammation and Light Touch Palpation, C. V. Mosby Co., St. Louis, 1912.

nerves themselves show a distinct pathology. Clinically, where the nerve is inflamed, it is accompanied by certain phenomena. Pain is a common accompaniment, although it may be so slight in character that it is only an uneasiness or a feeling of being tired. These sensations are expressly noticed through the shoulders and interscapular region and are nearly always confined to the side where the lung is involved. A pain of this character which I have not previously seen described is one which affects the pharynx. This, when present, nearly always comes on when the disease is far advanced. The patient complains of pain in the throat and yet nothing can be seen. These pains are entirely different from the sensory disturbances which have been described by Head. Head has shown segmental relationship between the reflexes of the lung and the skin, and in this has laid the foundation for the theory underlying the writer's observations. Every internal organ has a corresponding area on the surface of the body in which hyperalgesia may be reflexly developed when the organ is inflamed. This area is the one supplied by sensory nerves from the segments of the cord which are in communication with the affected organ through the sympathetics which supply it. The segments of the cord which stand in particular relationship to the lungs are the third, fourth and fifth cervical; and while pulmonary sensory reflexes may be found to take place with sensory spinal nerves from any of the cervical segments, these are the ones most commonly involved.

Not only do we have sensory, but also motor reflexes as I have shown. The spinal nerves in reflex communication with the lung show true degenerative inflammation. In this way I account for the instances of neuritis which I mention. Justification of the theory that these pains are due to injury to the neuron from reflex sources, is found in the clinical fact that the muscles supplied by the nerves from the segments in question take upon themselves increased tone or spasm when the pulmonary involvement is active, and degeneration when it quiets down, which indicates a serious nerve disturbance. In a former discussion the writer said:[10]

"While I do not wish to enter into a full discussion of the many pains that accompany intrathoracic inflammations (especially when found in tuberculosis) yet I cannot refrain from suggesting that their character indicates that some of them are phenomena of reflex origin. I am well aware that the theory of a reflex will not explain all, for we must recognize pleurisy, pressure pains, and general pains which are found in all parts of the body which may be of toxic origin. But, aside from these, we have regional or localized pains, the same as we have localized muscular contractions and atrophies, which must be explained by some locally acting cause. As a hint to explanation we find these localized pains for the most part, as far as I have been able to determine, on the same side on which the involvement exists. Thus it seems natural to suggest for them a common cause with other reflex phenomena.

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[10] Muscle Spasm and Degeneration in Intrathoracic Inflammation and Light Touch Palpation, C. V. Mosby Company, St. Louis, 1912.

"Of such regional pains the vague sensations which are found in the shoulder or shoulders in case of infection of both lungs must be mentioned. These often begin as early as clinical symptoms appear. They vary in intensity from a mere 'feeling of being tired' to an aching more or less severe. Aside from these vague feelings of discomfort we find pains of a very severe character which leave no doubt as to their nature. They are a true neuritis. These we find especially about the neck, shoulder (occasionally running into the arm), and upper portion of the chest both anteriorly and posteriorly. I have seen some instances in which the pain in these cases was so severe that it was necessary to resort to morphine injections. In some cases which I have observed there was a marked wasting with loss of muscular power. One thing characteristic of all less severe pains is that they are not constant. They come and go and are influenced by weather conditions and barometric changes.

"These cases have been misunderstood, wrongly diagnosed and consequently wrongly treated. They are quite often treated for rheumatism.

"The following cases will illustrate the severe forms:

"'Mrs. B. Age 32, had suffered from a slight infection of the right apex and an advanced tuberculous process of the upper left lung which had ended in cavity formation and healing. She had also had tuberculous ulceration in the interarytenoid space and left cord which resulted in healing. She suffered a great deal from aching of the left shoulder during her illness, and during the latter part of her treatment suffered severe pain running up the left side of the neck, which persisted, at intervals, for months, and then finally disappeared.

"Mr. L. consulted me in February, 1911, for a widespread ulcerative condition of the larynx. Upon examination of the chest I found an old fibroid lesion of the right lung occupying the upper half of the upper lobe, which was the seat of renewed activity. I told him my findings, that he had evidence of an old lesion in the lung. He protested that it could not be. I inquired as to his reason for leaving Philadelphia, which was his former home. He said he left because he had suffered from a severe rheumatism of the right shoulder which partially incapacitated him for business. I asked him if he was examined. He said he had consulted nearly a dozen of the best men there, all of whom had pronounced the case rheumatism. None had given him an idea that tuberculosis was present. During his last illness, for it proved fatal, he again suffered to some extent from the same pains. I have no doubt that his pains were due to a neuritis of reflex origin, the cause being the tuberculous process in the lung.

"Mr. G. consulted me in January, 1912, suffering from a pain in the upper part of the right chest which at first was considered to be of pleural origin. Owing to my inability to understand the patient's language, he being a foreigner, I was somewhat deceived for a few days. I found upon examination that the major portion of the upper lobe on the right side was involved in an old chronic tuberculous process and at the apex there were signs of cavity formation. His clinical history was interesting. He had always enjoyed good health, and, being a man of means, had lived well. In September, 1911, he was taken suddenly ill with chill followed by fever, severe cough, free expectoration and hemorrhages. The patient made a good recovery and had no further trouble until December, 1911, when the same thing occurred again. This was followed in about two weeks by the pain which I mentioned. The patient was admitted to the sanatorium and carefully watched; the pain persisted, coming on in severe paroxysms two or three times a day, sometimes requiring morphine. It lasted from a few minutes to one or two hours at a time. It is now nearly six months since it started and it seems to be gradually becoming less severe and less frequent. The pains seem to be confined to the brachial plexus, and particularly certain branches of it; viz., the dorsalis scapulæ supplying the rhomboidei, the thoracales anteriores supplying the pectoralis minor and major, the axillaris supplying the deltoideus and shoulder joint. Many of the other branches are also involved but these show the greatest pain. The muscles covering the anterior and posterior surface of the upper part of the chest, the shoulder and arm are all markedly degenerated and the strength of the right arm is very much reduced.

"It is very necessary to recognize these pains, for they offer suggestive diagnostic hints. Any of these pains, especially, if they are confined to one shoulder or the upper portion of the chest, call for careful examination of the lungs, to exclude a pulmonary involvement before any other diagnosis is made."

I wish to append the two following cases to further emphasize and illustrate the effect of tuberculous involvement of the lung in the production of brachial neuritis.

Case 2422.

Merchant, aged 35. Entered the Pottenger Sanatorium for Diseases of the Lungs and Throat, April 24, 1914.

Family History.—Mother died at 68, cause unknown. Father, living and well. One brother died of tuberculosis at the age of 21; two sisters died of tuberculosis at the respective ages of 23 and 30 years, all dying within a period of three months at the time the patient was ten years old. One was ill eight years, one three years, and one a year and a half.

Personal History.—The patient was never strong and was much under weight. He had the ordinary diseases of childhood with prompt convalescence. For many years had suffered periodically from severe hyperacidity and dilatation of the stomach, but no definite localized pain. Had vomited occasionally, the vomitus was never bloody.

Five years previous the patient had suffered an attack of pleurisy on the left side and on two or three occasions had raised small quantities of blood, the amount varying from a dram to a half ounce. The patient was examined at that time by a general practitioner and told that he had an old spot in the left lung. The patient gave this slight consideration.

Several times during the past five years this patient has suffered from hoarseness, which would last for several weeks at a time.

The present illness began about a year and a half prior to the time of consulting me, at which time the patient developed a persistent cough with some expectoration. A few months later his strength declined. He felt tired; his appetite began to fail; he also lost weight. Four months previously his throat became painful on swallowing, and was especially irritated by acids. Two weeks previously he suffered from attacks of pleurisy on the right side. At times he raised a small amount of blood. For four months past his voice had been decidedly husky most of the time. Had suffered from constipation for many years, apparently of the spastic type. Immediately prior to his entering the sanatorium he suffered from a very severe diarrhea of several days' duration.

He weighed 124½ pounds, his normal being 145 pounds. He slept fairly well and had no shortness of breath or night sweats.

Examination at the Time of Entrance.—His temperature was 99.4°, pulse 86, B. P. on the right side 115— on the left 95. Complexion, sallow; poorly nourished.

Physical examination of the chest showed severe infiltration of the upper two-thirds of the left lung, with a large cavity and diffuse infiltration throughout the entire left lung.

The right side showed moderate infiltration of the upper and middle lobes and of the apex of the lower lobe.

The larynx showed marked infiltration of the arytenoids and interarytenoid space and slight ulceration.

Brachial Neuritis.—Immediately prior to entering the institution the patient had some pain in the left shoulder which became very severe after he had been in the institution for a few days. The pain was constant, and affected the entire arm, but was most severe about the shoulder and upper arm. This pain was also accompanied by marked loss of strength which involved the entire arm. The pain was so severe that it was impossible for him to rest in any position and he was unable to sleep at night. The diagnosis of brachial neuritis was made.

It is interesting to know that this involved the same side as the severe involvement in the lung. After a month the pain gradually began to lessen and in six weeks' time it disappeared.

Case 2375.

Farmer, aged 51. Admitted to the Pottenger Sanatorium on November 18, 1913.

Family History.—Two brothers died of tuberculosis at the age of 21 and 23 years, respectively, at the time patient was about thirty years of age. Patient lived with brothers during their illness, but this was not the probable source of infection because, as will be seen, the patient had had pleurisy thirteen years before.

Tuberculous History.—At the age of eighteen the patient had a left-sided pleurisy. At the age of thirty-six developed cough with some expectoration and about the same time had a hemorrhage. Changed residence, going to some springs, where he improved markedly and from that time on had good health until 1908, when he contracted what seemed to be a cold; but it was accompanied by fever and a gradually increasing cough, and was followed by considerable loss in weight and strength. From that attack he never fully recovered and was unable to work up to the time ho consulted me. He spent eighteen months in a sanatorium in Iowa in 1912 and 1913. During his stay there he improved and at the time he was admitted to the Pottenger Sanatorium, he tired easily, had a capricious appetite, good digestion, a great deal of irritation in the larynx, followed by cough, some hoarseness, also some pain in the left shoulder. Temperature 99°, pulse 78, B. P. 110, weight 202 pounds, which was about a normal weight for a man of his size.

Physical Examination.—Physical examination revealed widespread disease in both lungs. In the upper portion of the left there was a suppurating cavity. The disease was not very active, but more of the fibroid type. In the right lung there was considerable activity, but without much breaking down.

On discharge from the sanatorium July 21, 1914, his temperature was 99°, pulse 82, weight 217 pounds. The condition in both lungs was markedly improved. There were a few scattered râles throughout the lungs but the principal involvement was confined to the area surrounding his cavity in the left lung. The patient was in good general condition, and able to take long walks without tiring.

Three months after his discharge from the sanatorium he consulted me again in my city office. At this time the condition in the lungs was about the same, his general condition was good, but he was suffering from a brachial neuritis on the left side. The pain was quite severe, preventing him from sleeping, and also interfering with his rest during the day. The muscles of the left arm gradually atrophied and the strength in the arm was very much reduced. This neuritis persisted for several months almost totally disabling the patient.

It is important to note that the brachial neuritis is on the same side as his severest involvement.

Pathology of Neuritis in the Tuberculous.—According to Hezel, the most common pathological change found in the nerves of the tuberculous is a parenchymatous degeneration. The nerve filaments degenerate and atrophy while the axis cylinder remains intact. In this early stage of degeneration no clinical symptoms are manifest. In the later stages of degeneration, not only the nerve filaments, but also the axis cylinder is involved and conduction is interfered with and later may be wholly lost. The sheath of Schwann and the endoneurium show certain alterations but nothing that can be considered as genuine inflammatory changes.

In certain nerves Wallerian degeneration takes place. In a few instances which have been critically examined the ganglion cells of the anterior horn as well as the special ganglia of those segments of the cord from which the nerves, which were the seat of neuritis, took their origin, showed histological changes in the way of different degrees of chromatolysis. These changes were more pronounced in the ganglion cells of the anterior horn than they were in the cells of the spinal ganglia.

Steinert[11] found degeneration in the ascending fibers of the spinal cord. The long fibers of the posterior column of the cord were degenerated. This was most marked in the cervical portion of the cord and confined to the posterior columns of Goll, the fibers of the posterior column which take their origin in the lumbar and sacral segments of the cord. The degeneration of the ganglion cells in Steinert's cases was in the sacral and lumbar portions of the cord, the portions from which the diseased peripheral nerves took their origin.

Jessen[12] in his splendid monograph and Hezel[13] in a later discussion cites the reports of many observers showing a varied clinic and pathology in nervous diseases, in which the tuberculous process itself seems to be the causative factor.

Jessen quotes Heine as finding degenerative changes in the phrenics in 27 of 29 patients examined and Japp as finding changes in the peripheral nerves of every one of 15 cases.

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[11] Zur Kenntnis der Polyneuritis der Tuberkulosen, Beiträge zur Klinik der Tuberkulose, vol. ii, 1904.

[12] Lungenschwindsucht und Nervensystem, Gustav Fischer, Jena, 1905.

[13] Tuberkulose und Nervensystem, in Brauer, Schröder und Blumenfeld, Handbuch der Tuberkulose, Bd. iv, 1914.

CHAPTER VII.

THE NERVOUS SYSTEM CONTINUED: THE VEGETATIVE NERVOUS SYSTEM IN ITS RELATIONSHIP TO DISEASES OF THE LUNGS: A DISCUSSION OF PRINCIPLES, INCLUDING THE ANTAGONISTIC ACTION WHICH IS MANIFESTED BETWEEN THE SYMPATHETIC AND PARASYMPATHETIC DIVISIONS.

Joint Chemico-physical Sensorimotor and Psychical Control.—The cells of the body are controlled by nerves and by chemical substances produced by the glands of internal secretion. So far as we know every body action must be produced through these channels. These two controls are brought into action by stimuli either of physical or psychical origin.

Many viscera produce secretions which have been given the name of internal secretions because they are not delivered from the gland through ducts, even though they are provided with them; but are contained in the venous blood as it emerges from the gland. These internal secretions have selective action; and, through them, one organ may be able to control or influence another organ far distant in the body. This may be illustrated by the action of adrenin, and the secretion of the thyroid gland, both of which exert influences upon cells and other structures throughout the body. Other glands produce important secretions which have a more selective action. All must work normally if harmonious physiological action is to be maintained throughout the body. A disturbance in one has a tendency to produce disturbance, either selective or general in others, the same as is produced reflexly through the nervous system.

It is impossible to definitely separate the chemical from the nervous control of the body cells. Owing to our paucity of knowledge, we are prevented from drawing absolute conclusions and putting the nervous and chemical control on an absolutely scientific basis.

In any study that is to be made of the vegetative nervous system, the clinician can only point out that such an action is the one that would be expected under a given condition. The resultant action, however, may be modified or prevented, by other factors, such as those produced through an antagonistic reflex, through chemical control, or through psychical or physical influences.

In this connection we must not forget the psychical influence over the human body. The subconscious mind is always active, not only influencing but determining our action and conduct, and also influencing our physiological activities. This important field is one that deserves further study, and promises to help us explain many conditions which cannot be explained on a purely physical basis.

The Vegetative Nervous System.—Vegetative, autonomic, and involuntary are all terms used to designate that portion of the nervous system which acts without the interposition of the will. This system furnishes impulses for carrying on the particular functions of the body which are necessary for the maintenance of life.

The writer has been greatly stimulated in this study by the works of Eppinger and Hess,[1] Walter B. Cannon,[2] Wm. H. Gaskell,[3] Heinrich Higier,[4] W. v. Bechterew,[5] S. C. Sherrington,[6] Lewandowsky,[7] Arthur Keith,[8] Biedl,[9] E. A. Schäfer,[10] Chas. E. de Sajous,[11] E. H. Starling,[12] W. M. Bayliss,[13] and contributions by Professor E. Gley, Nöel Patton, T. R. Elliot, and Swale Vincent, in the Practitioner's special number on "Internal Secretions."[14]

There is so much confusion in the terminology of this system and its divisions that it is necessary for each writer to make clear at the beginning of his discussion the meaning of the terms as he employs them. Throughout my discussion, I shall speak of the entire involuntary system as the *vegetative system;* and its divisions as the *sympathetic system*, comprising the motor cells which have pushed off from the thoracic and upper lumbar portions of the cord; and the *parasympathetic system*, comprising the motor cells which have pushed off from the cranial, bulbar, and sacral portions of the cord.

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[1] Die Vagotonie, Sammlung Klinischer Abhandlungen, von Noorden, Heft. 9 u. 10, 1910.

[2] Bodily Changes in Pain, Hunger, Fear, and Rage, D. Appleton & Co., New York, 1915.

[3] The Involuntary Nervous System, Longmans, Green & Co., New York, 1916.

[4] Vegetative or Visceral Neurology, Ergebnisse der Neurologie und Psychiatrie, Bd. ii, Heft 1, 1912.

[5] Die Funktionen der Nervencentra, vol. i, Gustav Fischer, Jena, 1908.

[6] The Integrative Action of the Nervous System, Charles Scribner & Sons, New York, 1906.

[7] Die Funktionen des zentralen Nervensystems, Gustav Fischer, Jena, 1907.

[8] Human Embryology and Morphology, Edward Arnold, London, 1913.

[9] Innere Sekretion, Urban und Schwartzenberg, Wien, 1910.

[10] The Endocrine Organs, Longmans, Green & Co., New York, 1916.

[11] The Internal Secretions and the Principles of Medicine, F. A. Davis and Company, 1903.

[12] Principles of Human Physiology, Lea and Febiger, 1915.

[13] Principles of General Physiology, Longmans, Green & Co., New York, 1915.

[14] Practitioner, London, 559, vol. xciv, 1 and 2, January, 1915.

I deem it unwise to follow the terminology of Eppinger and Hess in calling the latter group the "greater vagus" system even although all of the cells belonging to this group possess the property common to the vagus of antagonizing the sympathetics. The *parasympathetic* is often spoken of as the autonomic system (Jelliffe and White), in contradistinction to the sympathetic; but, inasmuch as this term is also applicable to the vegetative system as a whole, I think it advisable to refrain from applying it to one of the divisions.

In order to obtain a correct idea of the vegetative nervous system one must understand that the cells of this system have traveled out from the central nervous system. Very early in embryonic life before the neural folds have closed to form the spinal cord, these neuroblastic cells migrate. They are deposited here and there and form, for the most part, the ganglia of the vegetative system. Some of the neuroblastic substance, however, is deposited in other places, and this is known as chromaffin tissue; because, when stimulated, it secretes a material which stains brown with chrome salts. Physiologically, we know this secreted substance as adrenin or its immediate precursor.

The cells which migrate from those portions of the central nervous system which give origin to the parasympathetic fibers do not come to rest until they are situated in the walls of the viscera to be innervated by them. Here they form ganglia and throw out their processes which become motor and secretory fibers. They are connected with the central nervous system by connector fibers belonging to the parasympathetic system, as illustrated in Plate I from Gaskell, which run from the centers in the cranial, bulbar, and sacral portions of the central nervous system directly to the ganglia in the walls of the viscus supplied. There

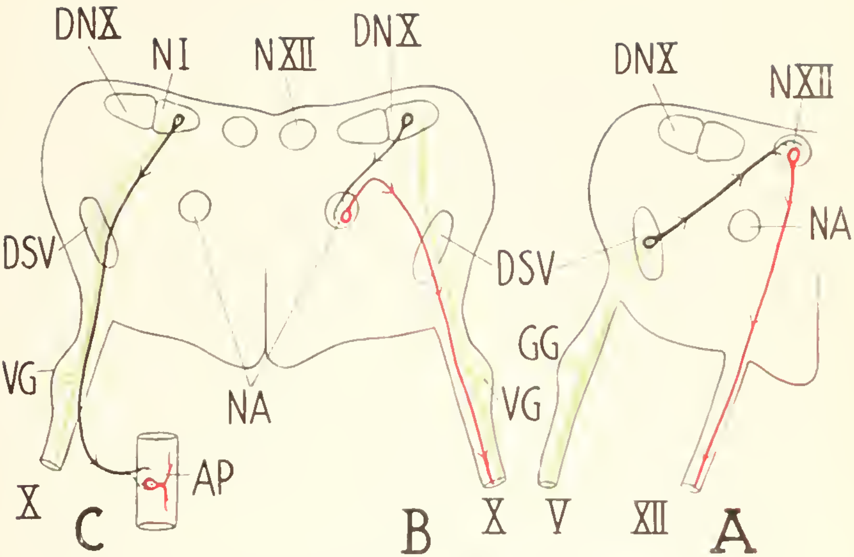

PLATE I.—THE REFLEX PATHS IN THE BULBAR REGION.

(*Green*—Sensory nerve; receptor neuron; *Black*—Connector neuron; *Red*—Motor nerve; excitor neuron.)

A. Of the Somatic System.

The afferent neurons run in the fifth nerve, *V*, their cells lying in the Gasserian ganglion, *GG*. These connect with the connector neurons lying close against the descending root of the fifth nerve, *DSV*. The connector neurons in their turn connect with the excitor cells which lie in the nucleus of the twelfth nerve, *NXII*.

B. Of the Splanchnic System.

The receptor neurons run in the tenth nerve, *X*, with their cells lying in the ganglion of this nerve, *VG*, and connect with connector neurons which lie in the dorsal nucleus of the vagus, *DNX*. Processes of the connector cells connect with the excitor neurons which lie in the nucleus ambiguus, *NA*, their processes form the motor part of the tenth nerve.

C. Of the Involuntary System.

The receptor neurons run in the tenth nerve, *X*, with their cells in the ganglion of this nerve, *VG*, and connect with connector neurons which lie in the nucleus intercalatus of Staderini, *NI*, which forms a part of the dorsal nucleus of the vagus, *DNX*. The processes of these connector neurons run out in the vagus nerve, *X*, and finally connect with the excitor neuron which lies on some peripheral organ; e.g., in the case of the intestine lying in Auerbach's plexus, *AP*.

(Gaskell.)

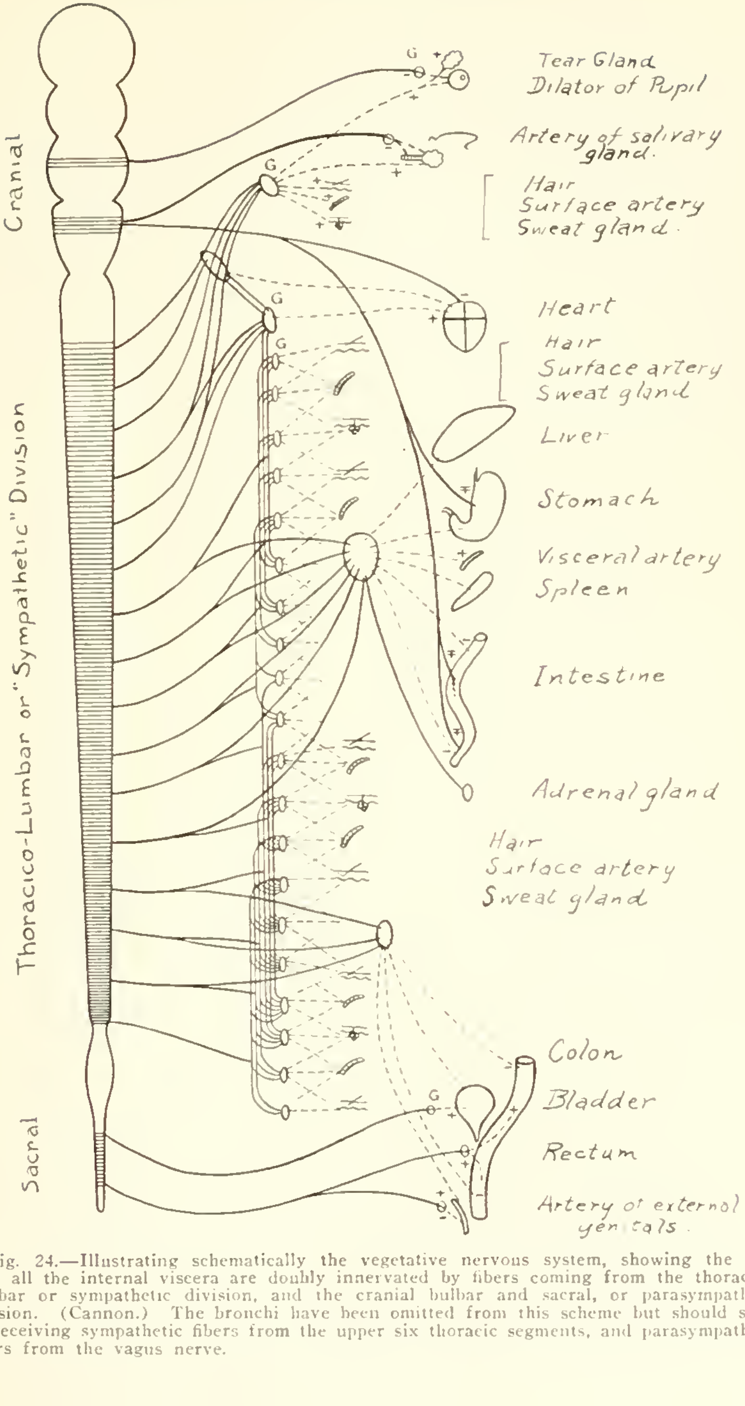

Fig. 24.—Illustrating schematically the vegetative nervous system, showing the fact that all the internal viscera are doubly innervated by fibers coming from the thoracico-lumbar or sympathetic division, and the cranial bulbar and sacral, or parasympathetic division. (Cannon.) The bronchi have been omitted from this scheme but should show as receiving sympathetic fibers from the upper six thoracic segments, and parasympathetic fibers from the vagus nerve.

are no ganglia on their paths. The scheme of the vegetative nervous system is shown in Fig. 24.

In the sympathetic system, the arrangement of these neuroblastic cells is different. As they wander out from the cord they are deposited in various places. First, there is a clump in front of each segment of the cord which, when joined together, make up the ganglionated cord of the sympathetic system. Then, there are other clumps here and there between the ganglionated cord and the viscera supplied. These clumps constitute the ganglia of the sympathetic nervous system. From them, fibers pass to form plexuses before going to the parts innervated. These ganglia are connected with each other so intimately as to make the stimulation of the sympathetic nervous system more or less general throughout all the parts supplied by it. The receptor neurons of the sympathetic system have their cell bodies in the posterior root ganglia the same as the spinal sensory. The sympathetic motor cells lie in ganglia outside of the cord, and are kept in connection with the cord by connector fibers which arise from cells in the lateral horn. This is illustrated in Plates

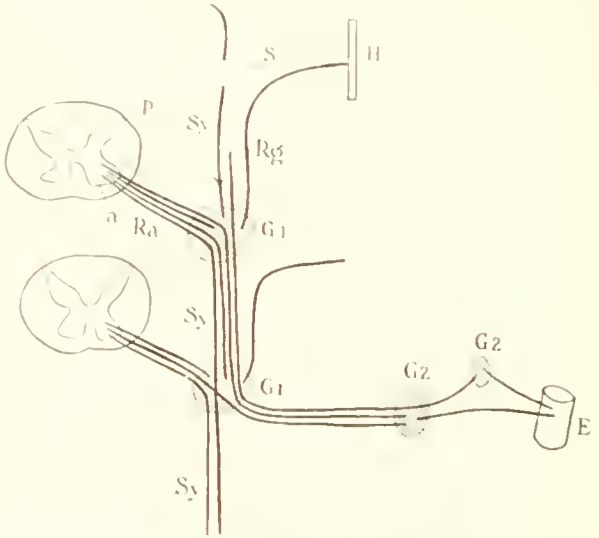

Fig. [illegible]. Illustrating schematically the course of the fibers in the sympathetic nervous system. *G1*, vertebral ganglion; *G2*, prevertebral sympathetic ganglion; *H*, skin; *E*, internal viscus; *a*, anterior root of the cord; *p*, posterior root of the cord; *S*, sensory nerve; *Ra*, ramus albus; *Rg*, ramus griseus; *Sy*, ganglionated cord of the sympathetic. (Lewandowsky.)

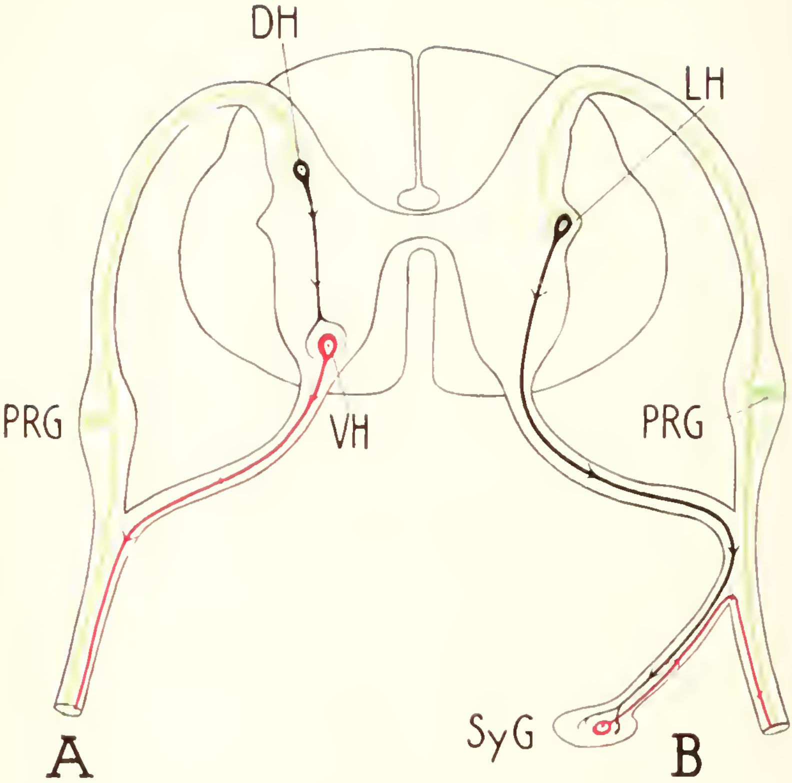

PLATE II. THE REFLEX PATHS IN THE CORD.

(*Green*—Sensory nerve; receptor neuron. *Black*—Connector neuron. *Red*—Motor nerve; excitor neuron.)

A Of the Voluntary System.

The receptor neurons run in the posterior root, their cells lying in the posterior root ganglion, *PRG*. The connector neurons lie in the dorsal horn, *DH*, and connect with the excitor neurons lying in the ventral horn, *VH*, whose processes run in the anterior root.

B. Of the Involuntary System.

The receptor neurons run in the posterior root, their cells lying in the posterior root ganglion, *PRG* The connector neurons lie in the lateral horn, *LH*, their processes running out in the anterior with the excitor neurons lying in the sympathetic ganglia, *SyG*. The processes of the excitor neurons form the gray ramus communicans and run out in the spinal nerve.

I and II from Gaskell. This whole sympathetic ganglionated system, however, is connected with the central nervous system by connector fibers, one going from each segment of the thoracic and upper lumbar portions of the cord to the ganglion of the ganglionated cord lying adjacent to it. It may connect with motor cells here or in more distal ganglia. The sympathetic filaments follow the blood vessels in their entrance to the organ. The scheme of the reflex path to the cord is shown in Fig. 25.

Those clumps of neuroblastic tissue given off from the thoracic and upper lumbar portions of the cord, which do not go to make up sympathetic ganglia, are deposited in various places and together make up the chromaffin system of the body. The principal point of chromaffin deposit in man is in the medulla of the suprarenal gland. It is also found at the branching of the carotids, and the branching of the aorta.

The portion of the chromaffin system which interests us most in our study is that found in the adrenal gland. It will be seen from this description of the development of the sympathetic system that the chromaffin tissue is an integral part of that system. This relationship is also shown physiologically. The secretion produced by it, adrenin, acts upon structures of the body supplied by the sympathetic nervous system, and produces the same action as though the sympathetic system was stimulated centrally. The action of adrenin, however, is exerted peripherally at the myo-neural junction in the organ innervated by the sympathetic system, instead of on central cells. The resemblance of the chromaffin tissue found in the medulla of the adrenal gland to the sympathetic ganglia is shown in another way. It has direct connector fibers with the cord, the same as sympathetic ganglia wherever they are found; consequently, every central stimulation of the sympathetic nervous system stimulates the adrenal gland, causing it to secrete adrenin. The stimulus passes directly to the gland, instead of passing through the collateral ganglia, as do the stimuli through the sympathetic system to the other internal viscera. In this respect its innervation is like that of the parasympathetic system; but it is unlike it because the system itself must be considered as a part of the sympathetic system.

The sympathetic cells come from the thoracic and upper lumbar segments of the cord only but go to every portion of the body. They supply not only every internal viscus, but the entire epidermal musculature, including the pilomotor muscles and the muscles of the sweat glands; and also furnish fibers which control the blood vessels throughout the entire body.

One connector fiber joins with many motor cells in sympathetic ganglia. In this way a central sympathetic stimulation is distributed widely peripherally; consequently central influences such as stimulation produced by toxins, and that produced by the depressive emotional states, like peripheral stimulation by such chemical substances as adrenin, act generally, influencing many of the body structures at the same time.

The parasympathetic system, however, is different in that its action is more selective. It is possible to have a stimulation which affects only the connector fibers between the central nervous system and the heart, or the stomach, or the eye, or the pelvic organs, leaving the others uninfluenced. In this system, however, if we find marked vagus stimulation in one organ, we are apt to find evidence of it in others.

The Inhibitory Action of Visceral Nerves.—The inhibitory action of the visceral nerves is not thoroughly understood and causes considerable confusion. It seems that when the vegetative system, originating motor nerves for the endodermal musculature, traveled out from the central nervous system, that it also, at the same time, gave off an inhibitory filament to opposing musculature. In many instances, from definite physiological experiment and discovery we cannot say that there are inhibitory nerves; but, from clinical observation of physiological action we know that such must be the case. There seem to be very widely distributed indications of reciprocal innervation between the parasympathetic and sympathetic systems. The motor cells of the parasympathetic, whether in the lungs or in the liver, or in the small or large intestine, lie in the muscles themselves, and their inhibitory nerves come from the cells of the sympathetic system. Other portions of the gastrointestinal tract, such as the esophagus and pharynx, offer probable exceptions to this. Some physiologists, however, claim that this same antagonistic action exists in the esophagus.

Grouping of Structures Supplied by the Sympathetic and Parasympathetic Systems.—Adrenin is a product of the chromaffin cells of the body which, in lower life, are closely associated with the cells of the sympathetic nervous system; consequently, in the higher animals, adrenin acts chemically, and produces the same effect as central stimulation of the sympathetic cells. Acetylcholine, on the other hand, produces the same effect when introduced into the body as stimulation of the parasympathetic division of the vegetative system; consequently, structures reacting to adrenin are considered as deriving their nerve supply from the sympathetic system, while those reacting to acetyl-choline derive their supply from the parasympathetic.

There is also a pharmacological test which is of great value in differentiating sympathetic and parasympathetic innervation. Atropin, when introduced into the body, counteracts the action produced by stimulation of the parasympathetics and produces the same effect as that which is produced by sympathetic stimulation. Pilocarpin, on the other hand, produces the same effect as stimulation of the parasympathetics; consequently, it counteracts the action of the sympathetics; so with adrenin and acetyl-choline; atropin and pilocarpin, we can go far toward determining which division of the vegetative nervous system produces a given symptom. In this connection I would mention that physiologically, we are led to believe that sweat is produced by sympathetic stimulation; but pharmacologically,—that is, by the use of atropin, we find that sweating is counteracted; and this would indicate that sweat secretion is a parasympathetic effect, rather than sympathetic. It is possible that it may be both.

The unstriped muscle of the body may be divided as follows, according to Gaskell.[15]

1. The vascular group. Every vessel throughout the body is supplied by the sympathetic nervous system. The nerves supplying the blood vessels take their origin from the entire sympathetic area, which extends from the first thoracic to the fourth lumbar segments. In every instance motor cells to the blood vessels of any organ are found in the ganglia which supplies the organ itself with sympathetic nerves.

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[15] The Involuntary Nervous System, Longmans, Green & Co., New York, 1916.

2. The group of muscles underlying the skin or epidermis. This includes the muscles to the sweat glands and the pilomotor muscles, which, when stimulated, cause erection of the hairs.

3. The group of muscles underlying the surface of the gut or endoderm.

4. The group of muscles around the segmental duct, which includes the Wolffian ducts, or urinary system, and the Müllerian ducts, or generative system.

5. The group of muscles forming that part of the gut wall, which particularly constitutes the sphincter muscles. Among these are included the *ileo-colic sphincter*, the *internal anal sphincter*, and the *internal vesicle sphincter*. The cardiac sphincter seems to be supplied by the vagus, while the activating nerve of the pylorus is in doubt.

6. The group of muscles connected with the adjustment of vision. These are the pupil, the ciliary body, the Müllerian muscle; and palpebral muscles.

Groups 1, 2, 4, and 5, are supplied by the sympathetic nervous system wholly, while groups 3 and 6 are supplied by the parasympathetic.

Aside from these particular divisions of the unstriped muscles of the body, all other internal viscera are supplied by fibers belonging to both the parasympathetic and the sympathetic divisions of the vegetative system. Wherever the sympathetic and the parasympathetic systems supply the same organ they are antagonistic to each other.

Symptoms Due to Stimulation of Vegetative Nerves are Variable.—It would give a wrong impression to indicate that we can with our present knowledge, follow out and accurately tell what symptoms will be produced by stimulating parasympathetic nerves, and what by sympathetic nerves; or tell what symptoms will be produced by this internal secretion, or that internal secretion; because other factors are active at the same time. We can only tell what the symptom would be if one factor were operating alone. Normally, we might have a stimulation of the vagus filament going to the heart, which would be sufficient to produce a slowing of the heart's action; but, there might be, counteracting this, either a central stimulation of the sympathetic system caused by toxemia, or a peripheral stimulation caused by adrenin or other internal secretions which act upon the sympathetics being thrown into the blood and irritating the sympathetic fibers which go to the heart, or an increased demand for oxygen on the part of the tissues which requires increased heart action, overcoming the slowing effect of the vagus. Or, I might cite as another illustration, a condition which we often see where a pulmonary tuberculosis is complicated by an intestinal infection. This complication is nearly always associated with considerable toxemia. Fever is a common symptom. Rapid heart's action should be an accompaniment of the toxemia. The heart's action, naturally, increases at a certain rate for a certain degree of toxemia, as indicated by the temperature curve; but where an organ supplied by the vagus is involved, there may be such a strong stimulation of the nerve endings of the vagus that a reflex decrease in the pulse rate is found. This often occurs in intestinal inflammation in spite of the fact that toxemia is accompanied by an increase of adrenal secretion; and, in spite of the fact that nearly all patients who have an intestinal involvement, suffer from more or less nerve depression, which, of itself, acts through the sympathetic, and has a further tendency to increase sympathetic stimulation. However, an understanding of the antagonistic action of the two divisions of the vegetative system, and an appreciation of the fact that the tonus of these two divisions differs in different individuals; and a further appreciation of the fact that both systems might be stimulated and yet the resultant action be due to the irritation of one of them predominating, will help greatly in the interpretation of symptoms.

Segmentation of the Body.—In order to understand the symptomatology of an infectious disease located in an internal organ, it is necessary to understand the nerve supply of that organ. In order to understand the nerve supply it is necessary to call upon our knowledge of embryology, and this brings us face to face with two of the most difficult subjects in physiology,—embryology and visceral neurology.

There are certain pains distant from the seat of inflammation which have long been recognized in medicine, such as pain in the shoulder, accompanying an inflammation of the central portion of the diaphragm, and pain in the left arm in diseases of the heart. Unless one has studied evolution from the lower forms of life, and the development of the human embryo, these symptoms are difficult to explain; but, when one is conversant with these subjects and understands the developmental relations, he can readily furnish an explanation for them.

In the early stages of evolution the body was made up of many similar segments joined together. In the simpler forms the segments were similar and had similar functions. As evolution proceeded, a differentiation of segments took place in order to form the different parts of the body. In the adult human body this differentiation is carried so far that it is necessary to return to the study of the embryo in order to grasp the similarity of the different segments (see Fig. 26).

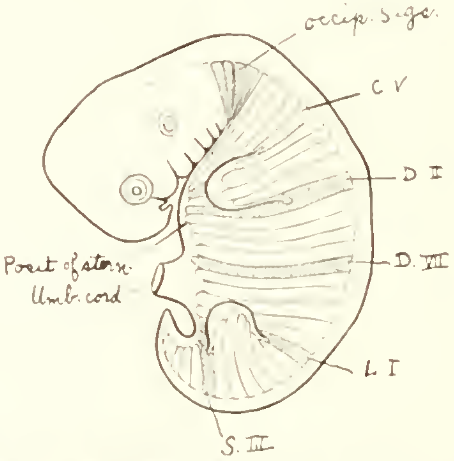

Fig. [illegible]. Diagram of a human embryo, fifth week, showing the arrangement and extension of the mesoblastic segments. The first and last of each segment entering into the formation of the limbs is stippled (*C V* to *D.II* and *L.I* to *S III*). The position is indicated in which the sternum is formed. (A. M. Patterson.)

The human body consists of thirty-three or thirty-four segments, fundamentally the same, but modified to form the cervical, thoracic, lumbar, sacral, and caudal regions of the body. These segments, or somites as they are called, consist of several structures: 1, skeletal; 2, muscular; 3, arterial; and 4, nervous.

It is an important fact, from the standpoint of the diagnostician to know that segmentation of the cord also takes place. From each segment of the thoracic and upper portions of the cord, four groups of cells arise: 1, somatic motor; 2, somatic sensory; 3, splanchnic motor; and 4, splanchnic sensory. The *somatic motor group* is situated in the anterior horn. It sends out processes to all the somatic muscles of the primitive segment in which it is situated. The *splanchnic motor cells* are found in the lateral horn and send out processes which reach the viscera belonging to the given segment through the white rami communicantes of the sympathetic system. The *somatic sensory group* is found in each segment. Its cells lie in the posterior root ganglia; and from within this, neuroblasts bifurcate and send out processes, one of which extends into the posterior horn, while the other goes to the skin, muscles, and subcutaneous tissue of the segment. The *splanchnic sensory group* is also situated in the posterior root ganglia and not in the ganglia of the sympathetic. This sensory group connects the internal viscera with the cord in such a manner that each viscus or part of a viscus is connected with some certain segment or segments of the cord.

These physiological facts relative to segmentation are of the utmost importance to clinicians; for whenever a part belonging to a body segment is diseased, the cells of the corresponding segment of the spinal cord are disturbed. This is of the greatest importance in the diagnosis of inflammation of the internal viscera; for *each viscus, or part of a viscus, is in reflex connection through the spinal cord with that area on the surface of the body which takes its nerve supply from the same segment of the cord.*

Segmental Relationship of the Lungs.—Head[16] pointed out that there were certain areas in the skin which showed trophic change and altered reaction to pain, heat, and cold, when the lung is inflamed.

The writer has called attention in numerous papers since 1909 to the spasm of the neck and chest muscles which occurs when the pulmonary parenchyma is acutely inflamed and to a degeneration of the muscles, subcutaneous tissue and skin which takes place when this inflammation becomes chronic, or heals after being present for a long time. The motor reflex which I have described clinically has not been described, to my knowledge, by the physiologist.

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[16] Brain, vol. xvi, 1893.

The lung is supplied by the sympathetic fibers from the upper six dorsal segments which proceed principally from the stellate ganglion. The stellate ganglion is made up of fibers from the upper three or four dorsal ganglia. The lung also receives vagus fibers coming from the bulbar roots. The fibers from the vagus supply the mucous glands and the musculature of the bronchus and produce increased secretion and increased muscular tone, when stimulated. The fibers of the sympathetic counteract the vagus and check secretion and produce relaxation of the bronchial walls and a reduction in bronchial secretion, when stimulated; consequently, such measures as will effect a sympathetic stimulation have a tendency to overcome bronchial asthma. This is the action of adrenalin and atropin. It also explains the observation which I have made in several cases where attacks of acute toxemia, through their stimulation of the sympathetics, relieved attacks of bronchial asthma. One of my patients was suffering severely from asthma, when he was given a dose of tuberculin which caused a toxic reaction. His asthma was slightly reduced in severity by the stimulation caused by the toxemia. The same patient had previously experienced freedom from asthma during a fever reaction caused by severe physical exertion.

The afferent impulses from the lung return to the upper five or six segments of the spinal cord, these being the segments from which the connector neurons for the lung arise; but, instead of mediating in the same segments with motor and sensory spinal nerves to form skeletal reflexes, the impulses are transmitted to intercalated neurons which carry them upward in the cord to the cervical segments, where they are transferred to the skeletal nerve. The segments which show the motor, sensory, and trophic reflexes most commonly are the third, fourth and fifth; although, when the irritation is severe, spinal nerves in other cervical segments may be affected. Thus it will be seen that there is an intimate connection between the lung and the cervical segments of the cord. Inflammation of the lung tissue, sending impulses centralwards, disturbs the nerve cells of this portion of the cord and causes motor, sensory, and trophic stimuli to pass peripherally to those parts which are innervated from these segments. Thus we have sensory disturbances more or less general throughout the cervical zones (Head), and motor disturbances throughout the somatic muscles, and trophic changes in all the tissues which take their innervation from this portion of the cord (Pottenger). While there are no sympathetic fibers which originate in the cervical portion of the cord, yet all cervical segments are connected with the afferent impulses which proceed from the lung.

Many writers have denied that the lung has sensation. From this we have been led to infer that there could be no reflexes in the somatic areas which are connected with the lung. This, however, has been shown to be untrue both by the work of Head and that of the writer.

Different segments of the cervical cord seem to receive impulses from the lung in varying degree. The point which seems to be particularly the center of bombardment is the third and fourth cervical segments. These segments supply nerve energy to the important muscles of the neck and chest including the diaphragm (all important muscles of respiration) and, through this fact, spasm of these muscles becomes an important symptom in inflammation of the lung. See table of muscles, page 330.

Head has also shown that the third and fourth cervical segments are the ones that are particularly prone to show the sensory disturbances when the lung is involved. We also have segmental sensory disturbances in the thoracic portions of the cord, in which the fourth and fifth thoracic zones are the chief centers of the impulse. For the location of these zones see Figs. 27 and 28, pages 182 and 183.

Lungs Embryologically Formed From Intestine.—In order to discuss the reflex symptoms which occur in pulmonary tuberculosis it is necessary to understand that the lungs were formed embryologically from a diverticulum of the intestine, consequently, they carry with them the same innervation as the intestine from which they are derived. They are thus closely associated with the gastrointestinal tract and show many reflexes in it. The reflexes, however, are not as common as those from one part

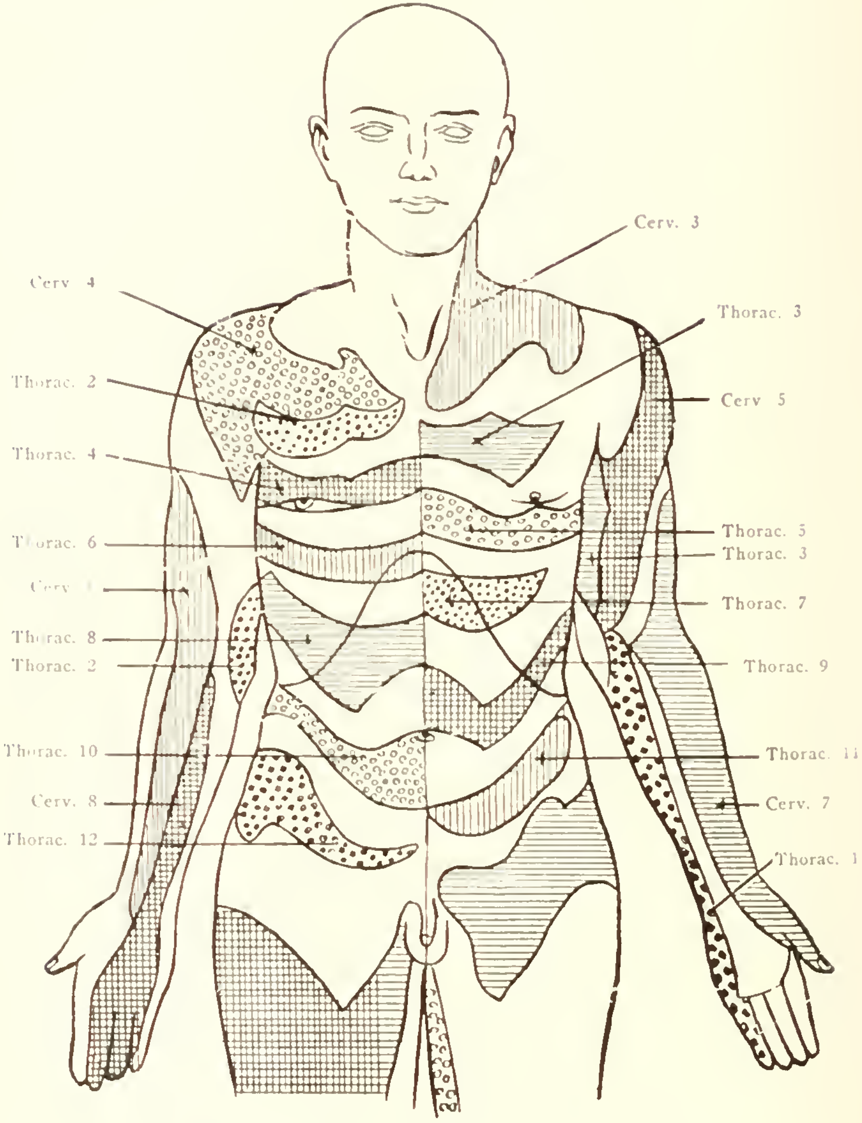

Fig. 27.—Head's zones. (Anterior surface of the chest and abdomen.)

of the gut to another. The motor cells of the parasympathetic system lie in the bronchial walls themselves and are connected with the medulla by connector fibers from the vagus.

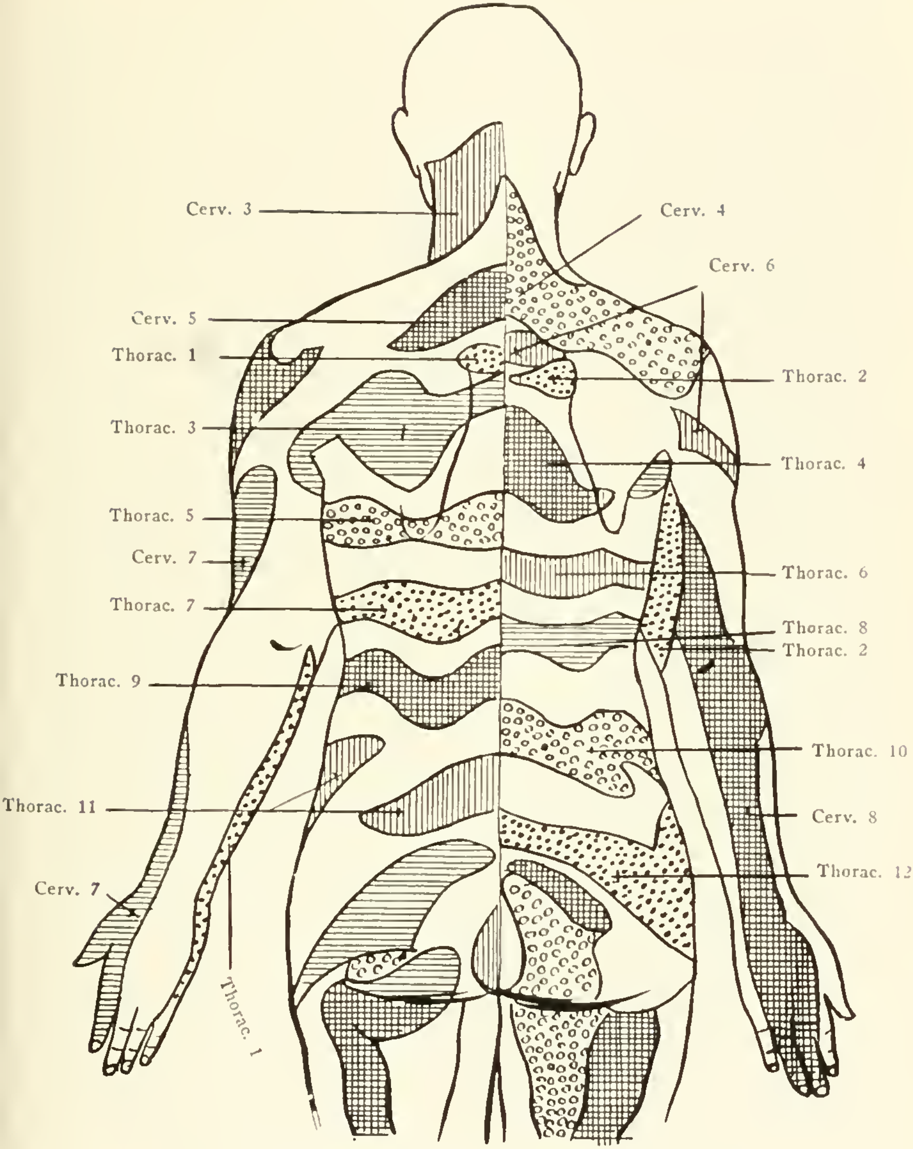

Fig. 28.—Head's zones. (Posterior surface of the body.)

The Relation of Symptoms in Tuberculosis to the Parasympathetic and Sympathetic Divisions of the Vegetative Nervous System.—In studying the clinical history and symptom-complex of pulmonary tuberculosis, everyone must have been more or less impressed with the indefiniteness of the symptoms and signs connected with this disease.

There are some twenty-five or thirty different symptoms which accompany early tuberculosis, and even more in advanced tuberculosis; and if we think of each symptom as an individual entity, there is no end of confusion. By carefully studying these various symptoms I have found that etiologically they belong to three groups, as shown on page 366. I first published this classification in 1914 [7] but I can see even more clearly than I did at that time the great value of this etiological study. By knowing the causes operating, we understand the reason for their variability.

This classification was arrived at after several years of study, during which time I was endeavoring to find an explanation for various reflex actions which were observed clinically. The study has been difficult because of the fact that its very foundation is surrounded by uncertainty. It was necessary, in many instances, to reason from effect to cause,—after noting the reflex action, to find its path. Later, however, after becoming more conversant with the vegetative nervous system and its action, I was able to determine that certain action in various structures should result from the stimulation of the pulmonary endings of the parasympathetic and sympathetic systems; and, in many instances, I have been able to find this to be the case.

The division of symptoms into groups, according to their etiology, has proved of great practical value in helping the examiner to weigh the importance of the symptoms which present in a given case; but there still remained other facts equally important to be determined. I then set for myself the task of going deeper into the etiology of these etiological groups to see if we could tell why toxemia produces malaise, a feeling of being run down, lack of endurance, loss of strength, nervous instability, digestive disturbances, loss of weight, increased pulse rate, night sweats, fever, and anemia; and how reflex action produces hoarseness, tickling in the larynx, cough, digestive disturbances, loss of weight, circulatory disturbances, chest and shoulder pains, flushing of the face, and apparent anemia; and why the symptoms of Group II are so variable.

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[7] Some Practical Points in the Diagnosis of Active Tuberculosis. Northwest Medicine, January, 1914.

The explanation to my first question came after and through finding the explanation for my second and third. I saw that the symptoms of Group II were due to action through two divisions of the vegetative system, the parasympathetic and the sympathetic: and, that their variability depended on the fact that these two divisions are antagonistic in action wherever they meet in the same organ and that the relative tonus of the two divisions differs in diffent individuals and at different times. I then saw that the symptoms on the part of the various viscera which were produced from stimulation of the sympathetics are the same as the visceral symptoms due to toxemia. This gave the clue that the toxic group of symptoms in tuberculosis is produced by stimulation of the central nerve cells but that it expresses itself peripherally through the sympathetics as an inhibition of function on the part of the internal viscera[18] supplied by both systems as dealt with more fully in Chapter VIII.

In this discussion I desire to treat at some length the subject of the antagonistic action between the parasympathetic and the sympathetic divisions of the vegetative nervous system, and show how this becomes a factor of great importance in determining not only the character of, but the presence of symptoms which are due either to central or peripheral stimulation of the pulmonary filaments of the vagus and sympathetic systems by the tuberculous inflammation in the lung.

The vegetative nervous system supplies impulses to, as previously mentioned, smooth muscles, such as the stomach, intestines, blood vessels, ducts of glands, skin, and secreting glands; also to certain organs possessing striated muscle fibers, such as the heart, the beginning and terminal portions of the alimentary canal, and the generative organs.

This system is characterized by the fact that under no circumstances do organs or parts receive innervation directly from a neuron whose cell bodies lie in the brain or spinal cord. Ganglia are interposed between the nerve cells in the central nervous system and the part innervated, which act as distributing stations for impulses. In these ganglia arise the true sympathetic neurons which carry impulses on toward the part innervated. These neurons are not under the control of the will. These outlying ganglia have preganglionic fibers which connect them with the cerebrospinal system, and postganglionic fibers which go to supply the viscera, as already mentioned.

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[18] Pottenger: The Early Pathological Changes and Their Relationship to Clinical Symptoms and Physical Signs in Pulmonary Tuberculosis, Southwest Journal of Medicine and Surgery, November, 1915; Clinical Symptoms and Physical Signs in the Early Diagnosis of Pulmonary Tuberculosis: A Discussion from the Standpoint of Their Etiology and Their Relationship to Each Other, Read Before the Robert Koch Society, Chicago, Illinois, October 18, 1915, Illinois Medical Journal, January, 1916; The Syndrome of Toxemia: An Expression of General Nervous Discharge Through the Sympathetic Nervous System, Journal American Medical Association, January 8, 1916.

The cerebrospinal nervous system, on the other hand, is controlled by the will. Its action is quick and definite. There are no ganglia interposed along the path of the nerve to modify the impulses which originate in the brain, but they are carried directly from cell bodies in the brain and cord to the muscles involved; and immediate action results.

The vegetative system consists of three divisions, the cranial, the thoracico-lumbar or sympathetic, and the sacral, as shown in Fig. 24 taken from Cannon. No connecting neurons for the vegetative system are given off from those portions of the cord which send out nerves to the fore and hind limbs.

The cranial fibers pass for the most part within the trunk of the *nervus oculomotorius* (third cranial) without interruption, to the ciliary ganglion, whence they furnish constrictor impulses to the sphincter of the iris. They also supply the ciliary muscle.

Bulbar fibers pass partly through the *nervus facialis* (seventh cranial) and *nervus glossopharyngeus* (ninth cranial) to the salivary glands, tongue, and blood vessels of the head. Stimulation causes increased salivary flow and dilatation of the vessels of the head.

The most important branch of the vegetative system coming from the bulbar portion of the cord is the *nervus vagus* which is the chief source of nerve supply for the internal viscera. It supplies the heart, bronchial tree, esophagus, stomach, intestines, pancreas and liver.

The sacral branch is the *nervus pelvicus.* It innervates the colon, sigmoid, anus, bladder, and generative organs. There is a divergence of view regarding the source of activating fibers for the colon which causes some confusion. Some physiologists state that the vagus supplies the gastrointestinal tract as far as the descending colon, and that the pelvic nerve supplies the remaining portion. Others, including Gaskell, state that the pelvic nerve supplies the entire colon.

Antagonistic Action of Parasympathetic and Sympathetic Neurons Shown in Variability of Symptoms.—Many structures are innervated by both the *parasympathetic* and *sympathetic* systems, and wherever this double innervation is found, the action of the two is antagonistic; as in the eye, where the parasympathetic contracts the pupil, the sympathetic dilates it; or in the heart, where the parasympathetic slows, the sympathetic accelerates. All vegetative organs of the body are supplied by both sympathetics and parasympathetics, with the exception of the sweat glands, pilomotor muscles, vascular muscles and the urogenito-dermal system which are supplied by sympathetics only and the ciliary body, esophagus and cardiac end of the stomach by the parasympathetics only.

The table from Eppinger and Hess[19] (page 188) shows this antagonism in the principal structures concerned in our study.

From it one can readily understand that the normal healthy state in the vegetative organs is produced and maintained by the sum total of sympathetic stimulation, equaling the sum total of parasympathetic stimulation. If either the one or the other overbalances, equilibrium is disturbed and dysfunction results.

Both the parasympathetic and the sympathetic divisions of the vegetative systems are influenced by a great number of natural as well as many pathological conditions.

These systems are influenced, in fact, regulated to a certain extent, during the state of health, by the secretions from the various internal glands. Each gland produces substances which contribute to the regulation of other glands and structures. These are called internal secretions. They are cast into the blood stream when formed and, circulating throughout the body, choose with precision the structure which they are to influence. Some, as those from the adrenals, pituitary and thyroid,

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[19] Vagotonia—A Clinical Study in Vegetative Neurology, Nervous and Mental Diseases Publishing Company, New York, 1915.

ACTION OF STIMULATION OF THE SYMPATHETICS	ACTION OF		ORGAN	ACTION OF		ACTION OF STIMULATION OF THE PARASYMPATHETICS
	ATROPIN	ADRENALIN		PILOCARPIN	ERGOTOXIN	
. . . . .	Para.		Sphincter iridis	Stim.		Stim. (N. III)
Stim. (Th. I-II)		Stim.	Dilator iridis			
. . . . .	Para		Ciliary muscle	Stim.		Stim. (N. III)
Stim. (Th. I-III)		Stim.	Orbital muscle	Para.	Para	
. . . . .	Para.	(Stim.?)	Salivary glands	Stim.		Ch.tympani secretion
Stim. (Th. II-IV)		Constriction	Cortical blood vessels			Dilatat. (N. X.)
. . . . .		Dilatation	Buccal blood vessels			Constrict. (N.IX.)
Constrict. (Th.II-IV)	Constrict.?	Constriction	Skin blood vessels, head region			
. . . . .	Dilatation	Dilatation	Coronary blood vessels	Constrict		
Constrict. (Th.II-L.IV)		Constriction	Intestinal blood vessels		Dilatat	
Constrict. (L.I-IV)		Constriction	Genital blood vessels			Dilatat. (N. pelv.)
Stim. (Th. II-L.IV)	Inhib.	Inhib.	Sweat glands	Stim.		
Stim. (Th. IV-VII)		Stim.	Philomotor muscles of the face		Para.	
Stim. (Th. I-V)	Stim.	Stim.	Heart muscle	Inhib.	Inhib.	Inhib. (N.X)
Relax (Th. II-V)	Relax.	Relax.	Esophagus	Stim.		Stim. (N.X)
Para. (Th.II-L.IV)	Para.	Para.	Cardia	Stim.		Stim. (N.X)
Dimin'd (Th.II-L.IV)	Diminished		Gastric tone	Increase		Increases (N.X)
Para. (Th.II-L.IV)	Para.	Para.	Gastric peristalsis	Increase		Increases (N.X)
Diminished?	Diminished	Diminished?	Gastric secretion	Increase		Increases (N.X)
Inhib. (Th.II-L.IV)	Para.	Para.	Small intestine peristalsis	Stim.		Stim. (N.X)
Relax (L. I-IV)	Relax.	Relax.	Colon	Stim.		Stim. (N.pelvicus)
Relax. (L.I-IV)	Relax.	Relax.	Sphincter ani muscle	Spasm		Spasm (N pelvicus)
Relax. (Th.II-L.IV)	Relax.	Relax.	Gall-bladder	Contract		Contract. (N X)
Inhib.?	Inhib.	Inhib.	Pancreatic secretion	Stim.		Stim. (N.X)
. . . . .	Inhib.	Inhib.	Bronchial muscle	Stim		Stim. (N.X)
Contract. (L.I-IV)		Contract.	Sphincter vesicae			Relax. (N.pelvicus)
Relax (L.I-IV)		Relax	Detrusor vesicae			Contract. (N.pelvicus)
. . . . .		Contract.	Uterus (Preg.)	Relax.		
. . . . .		Relax.	Uterus (Non.preg.)	Contract.		
Contract. (L.I-IV)		Contract.	M. retractor penis	. . . . .		Relax. (N.pelvicus)
Piqure		Raised	Carbohydrate tolerance	Diminished		. . . . .
Heat puncture		Raised	Heat balance	. . . . .		
Contract		Contract.	Pigment cells	Dilatat.		

Stim. = stimulation. Constrict. = constriction. Relax. = relaxation. Para. = paralysis. Inhib. = Inhibition.

act on the sympathetic nerves, while those from the pancreas and parathyroids, on the vagus, and still others on some particular branch of one or the other system.

Pathologically, toxemia produces a picture of central nerve stimulation, but a peripheral expression of general discharge through the sympathetic system. In inflammations of the internal viscera, whatever irritation is present, probably, as mentioned later, influences both parasympathetic and sympathetic filaments peripherally and causes them to stimulate structures reflexly.

It is interesting that slight toxemia, and the depressive emotional states such as fear, anxiety, discouragement, worry, and disappointment, are accompanied by the same symptom-complex. This is particularly easy to recognize in tuberculosis, because it is a disease which manifests so many periods of slight activity accompanied by a low or moderate degree of toxemia, which disappears after a few days' duration; and, because it is likewise accompanied by so many of the depressive emotional states, which come and go as the causes which produce them come and go.

The symptoms of toxemia and of the depressive emotional states are: headache, malaise, nervous irritability, insomnia, lack of appetite, coated tongue, inhibition of gastric and intestinal secretion and motility, constipation, fever, pallor, and, at times, sweating.

The above symptoms are not constantly present, nor do they manifest themselves in equal degree in each organ innervated by the sympathetic system. They are sufficiently constant, however, to indicate that, aside from general central stimulation there is a preponderance of sympathetic action peripherally.

The variability of the symptoms caused when the sympathetics are stimulated centrally in pulmonary tuberculosis is due to the fact that peripheral reflex stimulation of the pulmonary nerve endings of the parasympathetics is practically always present when activity in the pathological process is sufficiently acute to produce toxemia. It is possible; in fact, I have often observed that the reflex peripheral stimulation of the parasympathetics was sufficiently strong to overcome the central stimulation of the sympathetics. This I have seen manifested in one or more of the following ways: a moist clean tongue, a hyperacidity, or a relatively slow pulse, during periods of acute toxemia accompanying cavity formation in the lung.

After the acute activity passes over, unless the patient is prolonging the toxemia by wrong methods of living, particularly overexertion; or is suffering from such depressive influences as worry, fear, and discouragement, the central stimulation of the sympathetics lessens and whatever symptoms, arising from the nervous system, remain, are due to the irritation of the nerve endings by the inflammation in the lung, and are expressed as reflex phenomena in other viscera.

There is no question that peripheral stimulation of the parasympathetics, in one branch, will result in stimulation and the production of parasympathetic effects in other branches of the parasympathetics. Stimulation in the intestine causes slowing of the heart; and stimulation in the uterus, ovary or testicle causes vomiting; and, stimulation of the parasympathetics in the nasal mucous membrane will cause bronchial spasm or asthma.

Whether the same is true of the sympathetics is open to question. Sensory paths in the sympathetics are supposed to take the same course as in the spinal nerves. The nerve cells for them lie in the posterior root ganglia of the cord; and the path is supposed to be uninterrupted by sympathetic ganglia. Thus, Gaskell[20] says: "All the afferent fibers have their nutrient centers in the posterior root ganglia. No peculiarity, therefore, exists on the afferent side; the course of the sensory fibers is the same in all sensory nerves, viz.: direct to the cells of the posterior root ganglia, with no connection with any cells in sympathetic ganglia."

If this is true, then we are wrong in supposing that peripheral irritation of the sympathetic fibers in one viscus can cause reflex action in some other viscus except the impulse be carried to the cord, there to be transferred from the posterior root ganglion to the cells in the lateral horn and through the connector fibers to the motor cells in peripheral sympathetic ganglia, whence it is transmitted to the viscus. Bechterew[21] says: "The functions of the sympathetic nervous system are divided, as is well known, into sensory, motor, secretory, and trophic. The sensory sympathetic fibers transmit impressions to the spinal cord and brain; the motor, supply the involuntary or unstriped musculature. Aside from this, there are mixed fibers *which establish a connection between neighboring sympathetic ganglia.* The tonus of unstriped muscle fibers is reflexly maintained through sympathetic ganglia. The sympathetic system is also, without doubt, operative *in originating many reflexes in the sphere of the internal organs.*

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[20] The Involuntary Nervous System, Longmans, Green & Co., New York, 1916, p. 17.
[21] Die Functionen der Nervencentra, Gustav Fischer, Jena, 1908, pp. 59-60.

"*There is lacking today scarcely a single proof of the fact that cellular interruption of nerve fibers takes place in the sympathetic ganglia. With this fact established these ganglia assume at once the rôle of true nerve centers.* It follows as a consequence from the proof of interruption as produced by Ramon Y. Cajal that the nature and manner of the relationship between nerve fibers and nerve cells in the sphere of the sympathetic system is in reality the same as in the spinal and cerebral portions of the central nervous system."

Physiologists as a rule are opposed to this view expressed by Bechterew and insist that all reflexes which arise from stimulation of sensory sympathetic fibers must result from mediation in the spinal cord, the efferent impulse starting from the cord.

If this is true, then we must consider that the only way in which an inflammatory process in one viscus can influence another viscus through the sympathetics is by producing substances which gain entrance to the circulation and cause central stimulation of sympathetic cells as in case of the various toxemias, or peripheral stimulation as in the case of adrenin or, by the stimulus going back and mediating with efferent nerves in the cord.

In my discussion of the reflex symptoms found in tuberculosis and the antagonistic action manifested between the parasympathetics and sympathetics in their production, it will be necessary for the reader to bear in mind that there is grave doubt as to whether the sympathetics are able to produce a true reflex, which shows in other viscera without mediation in the spinal cord. There is no doubt, however, as to the influences which are present, stimulating the sympathetics centrally and increasing the general irritability of sympathetic cells. This at times overcomes the parasympathetics, and at other times is overcome by the parasympathetics. In my previous papers I followed the assumption of Bechterew that peripheral stimulation of the sympathetics may cause reflex action in other viscera; but, while clinical facts may seem to warrant this, it is best to consider it as still subject to doubt and most probably untrue.

In pulmonary tuberculosis after all conditions which effect general central or peripheral stimulation of the sympathetics seem to have been removed sometimes the tonus is greater in the vagus system, at other times in the sympathetic. As a rule, however, except during periods of increased toxemia, which I shall discuss later, my observation leads me to believe that the parasympathetic tonus is greater than the sympathetic in early tuberculosis. This is shown on the part of the heart during rest, and particularly on the part of the gastrointestinal tract. There are many exceptions to this, however, especially among those congenitally weak and those in whom sympathetic tonus predominates naturally.

In estimating the relative tonus of the parasympathetics and sympathetics it is necessary to bear in mind that increased tonus might show in one division of the nerves and not in all, and that this increased tonus in particular divisions might not be constant. This variability is due to the fact that there are many factors acting at the same time which cause stimulation and that some of these have selective action for certain structures. This is not only evident in the reflexes but also in the internal secretions which affect the two systems. It is also evident from the fact that the tuberculous patient may have increased or decreased tonus in either the parasympathetics or sympathetics from causes other than those which operate as a result of the pulmonary infection.

Because of the antagonistic action of the parasympathetics and sympathetics the function of many organs is rendered unstable when an unequal stimulation of one or the other occurs. The heart, while at rest, is often slower than normal, but on exertion, or during periods of toxemia or depression at once becomes more rapid than normal and settles down to the normal much slower than it should. The appetite, while disturbed during conditions which favor increased absorption of toxins such as exertion, or during periods of increased activity in the tuberculous foci, or during periods of depression and worry, is often normal and even above normal when the patient is put at rest in the open air under favorable circumstances which offer him hope of cure. The same is true of the gastric and intestinal functions. Hyperacidity and hypermotility are common in early tuberculosis, when toxemia is relieved. The intestinal tract also shows the same increased tonus. The parasympathetic system, which if slightly in the ascendency, conserves the healthful action of the important enteral organs, overcomes its antagonist in early tuberculosis for the most of the time and this offers the patient an increased opportunity for restoration to health. If parasympathetic tonus is excessive, however, it proves harmful.

During waves of increased activity in the tuberculous focus, however, and during the period when the patient is causing increased absorption of toxins by overexertion and other faulty habits, likewise during periods of great depression from other causes, the sympathetic system is excessively stimulated both directly and by internal secretions and overcomes the increased parasympathetic tonus and causes symptoms which are characteristic of sympathetic irritation, such as sweating, rapid heart, decreased appetite, hypochlorhydria, deficient gastric motility, and alteration in the secretion and motility of the entire intestinal tract.

Aside from the antagonistic action of these two systems antagonistic fibers are at times supplied to organs by the same system, and action and inhibition depend on different degrees of stimulation.

In health a state of equilibrium is maintained in the various organs as a result of the antagonistic action of these two systems. In any disease which affects either by stimulation or by setting aside the normal nerve tonus of any important branch or group of fibers of either or both of these systems, there is a consequent disturbance of equilibrium which results in a pathological state.

This is evident in the production of nausea through eye strain, or the slowing of the heart in abdominal lesions which affect the depressor fibers of the vagus. So it is evident in pulmonary inflammation.

Effect of Internal Secretions on Symptomatology.—Aside from the direct stimulation of the nerves by inflammatory processes we must recognize the influence of substances of a chemical nature which act either upon terminal filaments or nerve centers. Thus the primary action of toxins is upon the central cell bodies. A secondary action may be a totally different one and depend on certain secretions which are set free as a result of the primary toxic influence, such as occurs when adrenin is set free by certain emotional states or gastric secretion is stimulated by the smell or taste of savory food. No doubt future study will clarify this subject very much by the discovery of the true relationship which exists between many of the internal secretions and the structures controlled by them.

Internal Secretion of the Thyroid.—At this point of our discussion it might be well to call attention to the enlargement of the thyroid gland, which occurs commonly during early clinical tuberculosis. The thyroid is the energy controlling gland of the body. Belonging to the sympathetic system, it is stimulated by the same factors which stimulate the sympathetic nerves such as toxins and emotions, which call upon the body for active defense. I have often noted that a hyperactivity of the thyroid gland takes place in the presence of toxemia in early tuberculosis. It also occurs in other toxemias such as those arising from infected tonsils and sinuses and from the intestinal tract. When toxemia is present, as I have discussed throughout this monograph, there is a general sympathetic stimulation. One of the results of sympathetic stimulation is increased action of the adrenal gland. Adrenin stimulates the thyroid, so it is natural to suppose that increased adrenin calls for increased flow of thyroid secretion. It is a principle in physiology that increased function, not only of muscles, but of secreting organs, is accompanied by increased blood to the part, and increase in the size of the cellular components; consequently, it is not at all beyond reason to suppose that these enlargements of the thyroid gland, which occur in early clinical tuberculosis, and which are also noted when we have infections in other parts of the body, such as the tonsil and nasal sinuses, might be due to a hyperactivity of the gland, resulting from increased stimulation. That this explanation is plausible is further implied in the fact that the gland returns to normal after the toxemia disappears.

Internal Secretion of the Ovary.—It is also well to bear in mind the influence of the secretion of the corpus luteum in the production of the premenstrual rise in temperature. I have suggested, as an explanation of this phenomenon, a stimulation of the sympathetic system, which, acting through the vasomotor center causes vasoconstriction and interference with the dissipation of heat (see Chapter XXX). These nervous phenomena which occur at the menopause when the ovarian secretion ceases are undoubtedly largely those of disturbed nerve balance, and particularly parasympathetic hyper-irritability; and further indicate that the secretion of the corpus luteum acts by stimulating the sympathetic nervous system. The premenstrual nervous disturbances which so commonly complicate our symptom-complex in tuberculosis become more intelligible with the understanding of their causation.

Symptoms Appearing at the Menopause Due to the Cessation of Ovarian Secretion.—The menopause is usually considered as a critical period in woman's life. It is a time when the normal resistance seems lessened, therefore it is of great interest to the students of tuberculosis. It has long been known that at that time a general atrophy of the female generative organs takes place; but the manner in which this acts to produce the symptoms noted has not been thoroughly understood.

At this time instability of the nervous system is the most prominent symptom. With more accurate clinical observation and our increasing knowledge of the action of the glands of internal secretion, this condition becomes more understandable. As puberty comes on, ushering in the child-bearing period, very marked changes take place in both the physical and nervous make-up of the individual. In this new period of woman's life there is one particular new force added to those which have been previously operative; namely, the internal secretion produced by the sex glands, particularly the ovary. The secretion of the corpus luteum acts upon the sympathetic nervous system and stimulates all structures supplied by it. Consequently there is a stimulation of certain other glands of internal secretion,—such as the adrenal gland, stimulation of which causes increased oxidation; and the thyroid gland, stimulation of which causes increased metabolical activity. The action upon these two glands of internal secretion is most evident, clinically, but doubtless the influence of this secretion is felt generally; because we cannot conceive of any new potent force coming into the chain of internal secretions without disturbing the established equilibrium in all.

The disturbance of this equilibrium is not continuous during the child-bearing period, but is most marked during the period of ovulation. This causes the cycle in woman's life which has been clinically observed throughout the child-bearing period. It would seem that this ovarian secretion is given into the blood stream irregularly even in the same individual and that its influence upon the system is much greater in certain individuals than in others.

In observing many cases of pulmonary tuberculosis over prolonged periods of time, I have noticed the influence of this secretion as it expresses itself in the nervous system. The manner in which it influences the pulse and temperature is shown in clinical charts which are given throughout these pages, and may be particularly studied in patients whose temperature and pulse curves are shown over a long period of time in "The Tuberculosis Clinic," Chapter L, Vol. II.

It will be noticed that in some patients this influence shows with almost absolute regularity month after month. It may be possible that this internal secretion of the ovary is given off continuously; but it is given off in greater quantities in most women during the two weeks preceding menstruation. At times its influence is seen on the 14th day following menstruation and shows as a slight rise in temperature, which continues until the day of menstruation when it drops again. Sometimes the pulse will also show an acceleration. In others this may not be noticeable. My observation has been that acceleration of the pulse is not as general as the elevation of temperature, but that it occurs most commonly when marked nerve irritability is present. Menstruation is also preceded by a rise in blood pressure, and accompanied by a fall in pressure.

When marked nerve stimulation is present, it manifests itself in such symptoms as insomnia, depression, disturbed appetite and increased irritability. These symptoms are more marked as a rule only a day or two preceding menstruation. According to my observation, this pre-menstrual rise in temperature may come on any time from the 14th day prior, up to the day of menstruation itself. In a few instances it would seem that this ovarian secretion does not affect the patient prior to menstruation but only during the same and probably for a few days following. In these cases there seems to be a reverse curve. This will be noticed in some of the charts in these pages (see Fig. 128, Vol. II) ; but this is infrequent. We assume that the stimulation of the sympathetic is coincident with ovulation, and are fortified in this assumption by the fact that these symptoms of sympathetic stimulation disappear when the menopause has been passed.

During the child-bearing period the organism gradually accustoms itself to the ovarian secretion and its stimulating effect upon the nervous system. The effect of any sympathetic stimulation if counterbalanced at all, is counterbalanced through stimulation of the parasympathetics. It is probable that in many individuals this ovarian secretion is so slight in its influence on the nervous system that its action is within the bounds of balance; but where the nervous stability is threatened by the influence of the ovarian secretion, equilibrium may be maintained by some natural physiological antagonist to sympathetic action.

When the necessity for further ovulation ceases, this ovarian secretion also ceases. The result is a marked unbalancing in the nervous system. The picture on the part of the vegetative system is that of marked instability, parasympathetic tonus, for the time, being in the ascendency. Not only the vegetative nervous system but the central nervous system and psychical control are often disturbed. So this is a period when woman is required to adjust herself to new conditions.

Our conception of the etiology of symptoms which accompany the menopause, are:

1. A withdrawal of ovarian secretion from circulation.

2. The elimination of the normal stimulation of the sympathetics produced by the ovarian secretion, which results in a disturbance of balance in the vegetative nervous system, permitting parasympathetic tonus for the time being to be in the ascendency.

3. A disturbance on the part of the higher nerve centers as a result of the general disturbance in the vegetative nerve and endocrine systems; and possibly also directly as a result of the absence of the action of the ovarian secretion on the higher centers.

4. A general psychical instability resulting from the heightened irritability of the vegetative system and the higher nerve centers.

ANTAGONISTIC ACTION OF PARASYMPATHETICS AND SYMPATHETICS AS SHOWN IN SYMPTOMS OF PULMONARY TUBERCULOSIS

Let us now proceed to apply the principles involved in the stimulation of the nerves of the vegetative system to pulmonary tuberculosis and the explanation of such phenomena as are caused by the disturbed equilibrium in the two systems.

In tuberculosis we must conceive of a condition in which both the parasympathetic and sympathetic systems are at times simultaneously stimulated, and one in which either one or the other may yield with a resultant disturbed equilibrium.

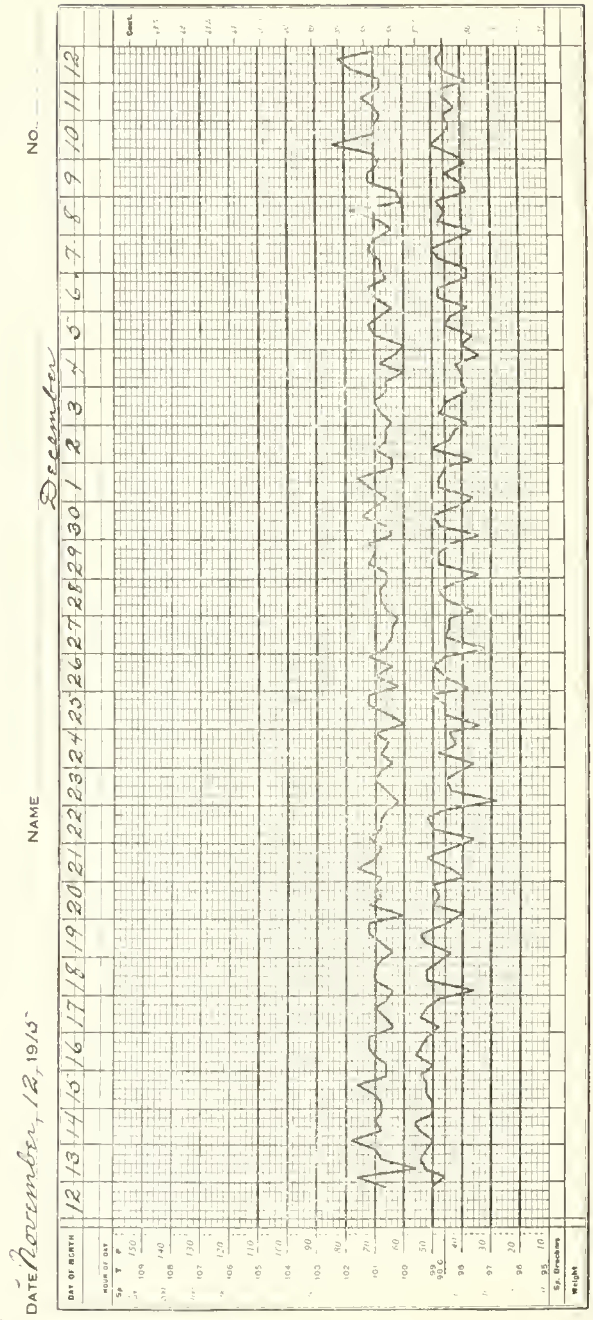
Dilated Pupil.—At least 50 per cent of tuberculous patients show a dilated pupil on the side of the involvement during some time when the inflammation is acute; and it is probable that a much larger per cent would show it if observation could be made continuously. This indicates that the action of the sympathetics overcomes the parasympathetics. This dilator effect is due to irritation of fibers originating in the upper thoracic segments, the stimulus being either central or reflex, coursing over the sympathetics in both directions. Aside from the disturbances in the pupil the ciliary body is stimulated to contractions by irritation of the parasympathetics. I am inclined to believe that there is often a disturbance of accommodation in tuberculous patients due to the unequal reflex stimulation of the pulmonary ends of the vagus. It is surprising to see how many patients complain of headache if they continue to use their eyes for reading, knitting, or sewing, as they have been accustomed to do prior to their illness. They also seem to show an increased sensitiveness to bright light and I often find it necessary to suggest that the bed be so placed that the light will not shine directly in the eyes. Change of eye glasses is made necessary more often than prior to illness even in early and chronic cases which are only slightly active and the eyes are more difficult to fit.

Hectic flush has generally been considered as being due to toxemia; but, being a one sided phenomenon and found on the side of the pulmonary involvement where one lung only is affected; or, on both sides, but varying in degree according to the degree of activity, where both lungs are affected; it seems that reflex action alone can explain it.

It has been shown that stimulation of sensory spinal roots either before or after they have passed through the posterior root ganglia causes dilatation of the vessels in the area supplied by the roots, no matter what the character of the stimulus. The sensory fibers of the fifth cranial nerve are analogous in their function to the sensory fibers of the spinal nerves; and they stand in the same relationship to the afferent fibers of the vagus in the production of reflexes as the spinal sensory nerves do to the afferent fibers of the sympathetic system. It has further been shown that there are dilator fibers in the trigeminus which are of cerebral origin. Therefore the vagus and trigeminus are nerves which carry respective afferent and efferent impulses and offer a ready explanation for the causation of hectic flush. Dilatation of the vessels of the ear, on the other hand, is through the sympathetics and third cervical nerve.

Heart.—In the heart in tuberculosis we have so many conditions present which influence the pulse rate that it is impossible to accurately ascribe to the parasympathetics and sympathetics the part which each plays. Impulses are carried through both systems, because both are constantly irritated.

As a result of this double source of impulses, the one through the vagus tending to slow the heart, the other through the sympathetics, attempting to quicken its action, there is a marked



Sp—Sputum

Fig. 29.—Illustrating predominant parasympathetic tonus as shown in slowing of pulse in a patient suffering from moderately extensive pulmonary tuberculosis.

disturbance of equilibrium. This shows early in the disease before such conditions as loss of pulmonary tissue, heart strain, and degenerative changes can be considered. The heart beat may be normal, slower than normal, or only slightly faster than normal while the patient is at rest; but it is often unduly rapid on exertion, and returns to normal slower than in the healthy individual.

This relative slowness of the heart beat is often noticed during periods of temperature, as compared with the same degree of temperature in other diseases. This is unquestionably the result of an inhibitory reflex through the vagus, the impulse coming from the irritation in the lung. When the intestinal tract is affected with tuberculosis, then another division of the parasympathetic system is stimulated and we often see this inhibitory action accentuated, with still greater departure in pulse rate from that which would be expected with the degree of temperature present. If an unusual slowing of the pulse occurs in the course of pulmonary tuberculosis, coincident with an elevation of one or two degrees in the temperature curve, reflex vagus irritation should be considered as a probable cause and a complicating intestinal tuberculosis be suspected.

These vagus influences are seen very often clinically, as shown in the following charts:

Fig. 29 is the chart of a patient suffering from moderately advanced tuberculosis, but in whom toxemia was a very small factor. It will be noted that the temperature which was between 99° and 100°, on entering the institution dropped to practically normal after 10 days' rest. The pulse was found, as a rule, between 60 and 70. This patient's resisting power was good and his chances of recovery splendid, in spite of a widespread lesion. This patient was markedly vagotonic.

Fig. 30 shows the pulse of a patient suffering from very extensive inflammation of the right lung, which went on to the rapid formation of fibrosis, and eventually resulted in arrestment. This case is described in Volume II, Chapter L, Case 2,514. It will be noticed that while the temperature was ranging above 102° the pulse was not exceeding 100 beats per minute. Toward the end of the month a very interesting condition presented it-

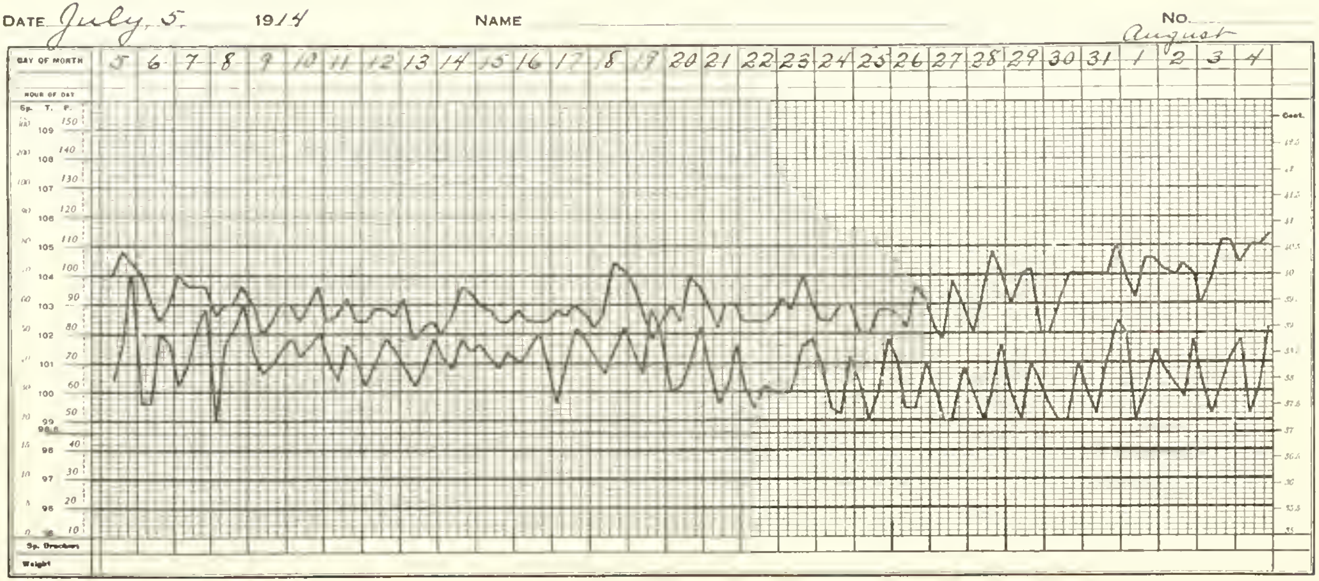


Fig. 30.—Showing pulse rates low in comparison with the temperature curve, indicating the predominance of vagus stimulation. The rise of the pulse during the last week of the month coincident with a fall in temperature indicates a relief from vagus stimulation. The pulse assumes the normal maximum for the degree of temperature present.

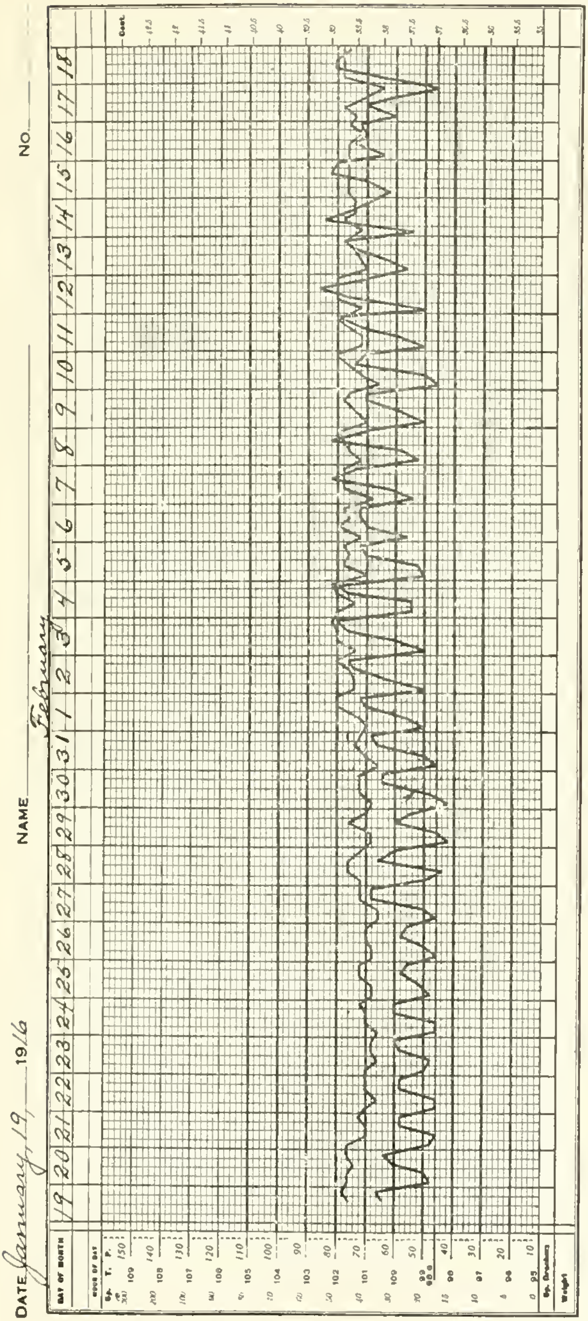


Fig. 31.—Showing a temperature curve of acute caseous pneumonic phthisis with relatively low pulse, the parasympathetic stimulation predominating over the sympathetic.

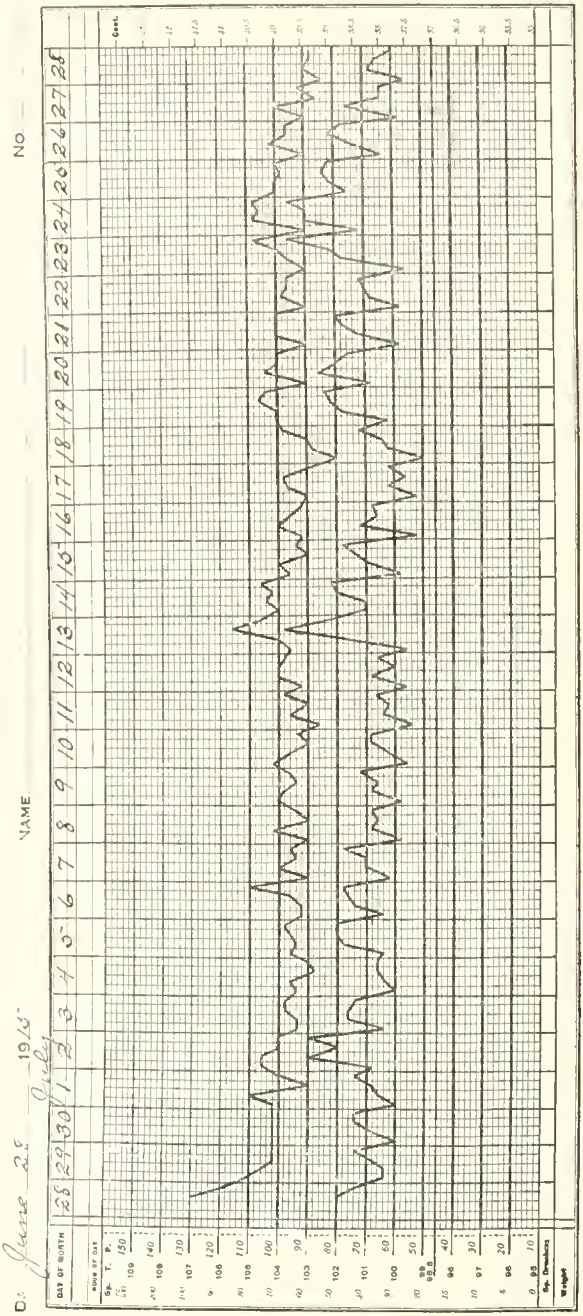


Fig. 32A.—Illustrating pulse and temperature curve of a patient suffering from acute caseous tuberculosis. The pulse rate is slower than normal for the degree of toxemia present, as manifested by the temperature curve. This is due to reflex vagus stimulation overcoming sympathetic tonus.

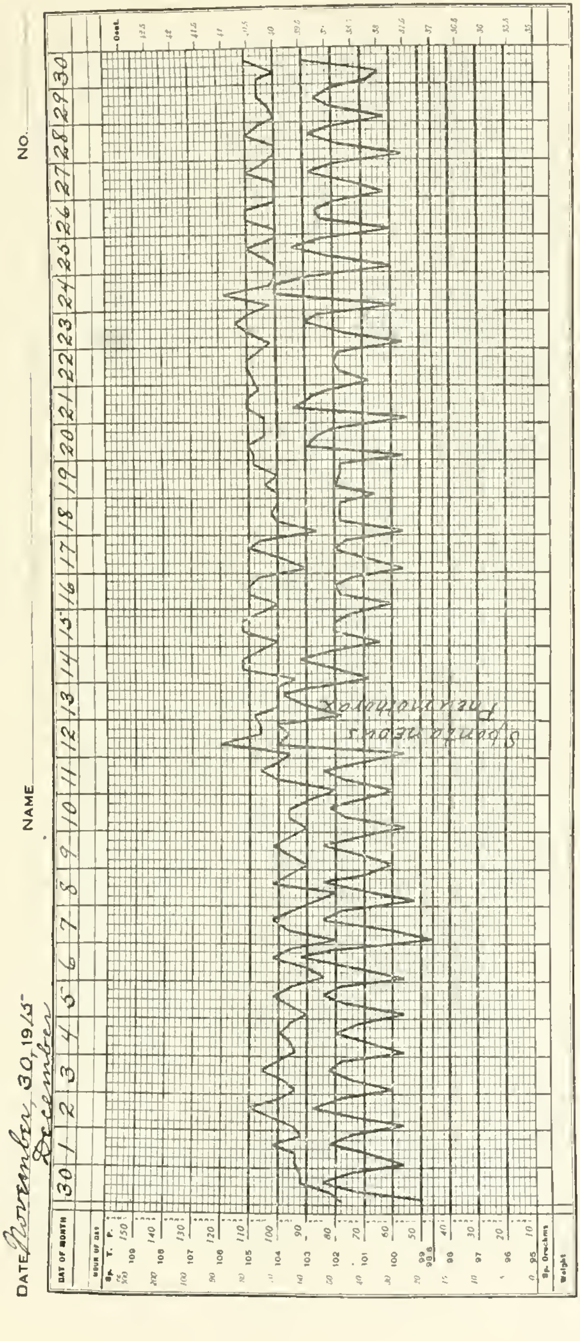


Fig. 32*B*.—Illustrating the same as Fig. 32*A*. The increase of temperature and pulse rate on the 12th of the month was due to spontaneous pneumothorax.

self,—the temperature dropped and the pulse increased in frequency. This may be explained thus: as the inflammation in the lung lessened, the toxemia decreased and the temperature dropped and the pulse should have become still slower, but the reduced inflammation also lessened the amount of irritation of the pulmonary vagus and permitted the antagonistic action of the sympathetic to increase the pulse rate. The patient was in bed not even sitting up to eat during the entire month.

Fig. 31 shows the temperature curve of a patient suffering from acute caseous pneumonic phthisis. It will be noticed that the disproportion between the temperature and pulse was very marked. While the temperature ranged from a maximum of 100° to 102°, the pulse remained between 70 and 80. This patient eventually died of miliary tuberculosis, with meningeal complication, although there was no clinical evidence of either intestinal or meningeal infection present to cause the slowing of the pulse at the time represented by the chart. See page 203.

Figs. 32 *A* and *B* illustrate the disproportion between the temperature and pulse rate in a patient with acute caseous tuberculosis which later went on to death, but was preceded by marked intestinal disturbances. It will be noticed that there is a disproportion between the temperature and pulse,—the pulse being comparatively slow, considering the serious condition of the patient. It must further be said that this patient's pulse was running in the neighborhood of 120 and 130 prior to his entering the institution; but, when put at rest, it dropped markedly and continued between 90 and 100 most of the time. The increased rapidity shown in chart *B*, beginning on the 12th of December, was due to spontaneous pneumothorax. This rise in temperature and pulse was due to a limited pneumothorax, but the extra disturbance was an added factor in raising the pulse during the latter half of December. It was very interesting to note that with the increase in inflammation in the lung shown in the first half of chart *B* there was not a corresponding increase in pulse rapidity. The fact that a spontaneous pneumothorax followed this increased inflammation on the 12th of the month suggests that the cause of the increased temperature was to be found in the activity of the tuberculous process in the lung; consequently,

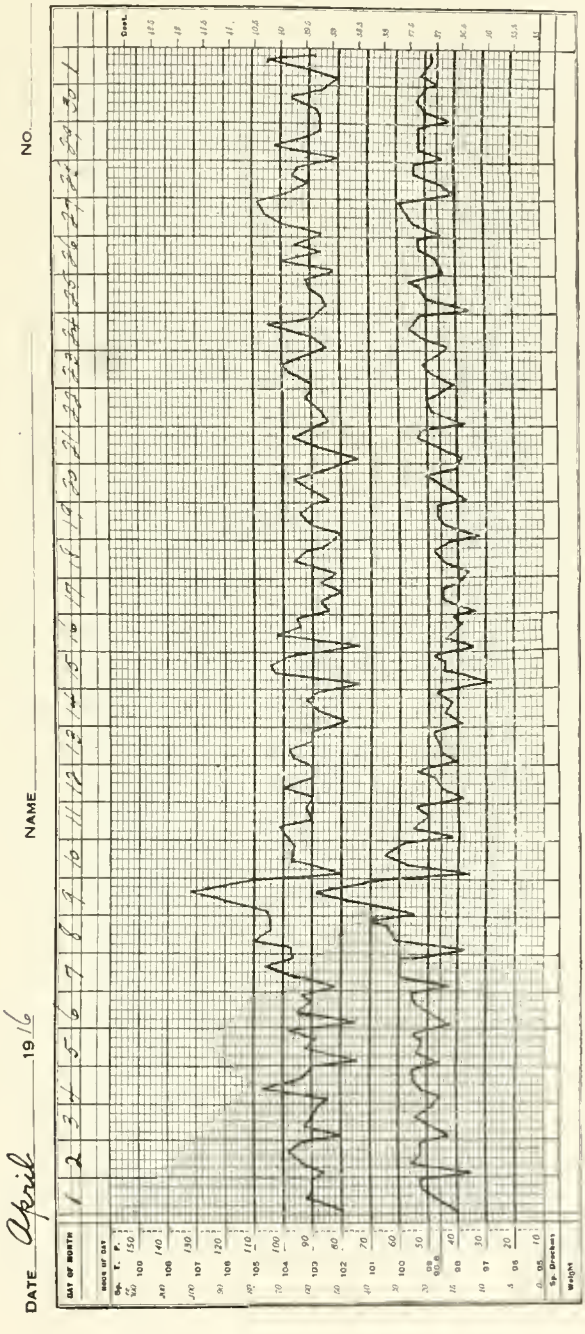


Fig. 33.—Illustrating the manner in which the pulse and temperature follow each other during periods of toxemia.

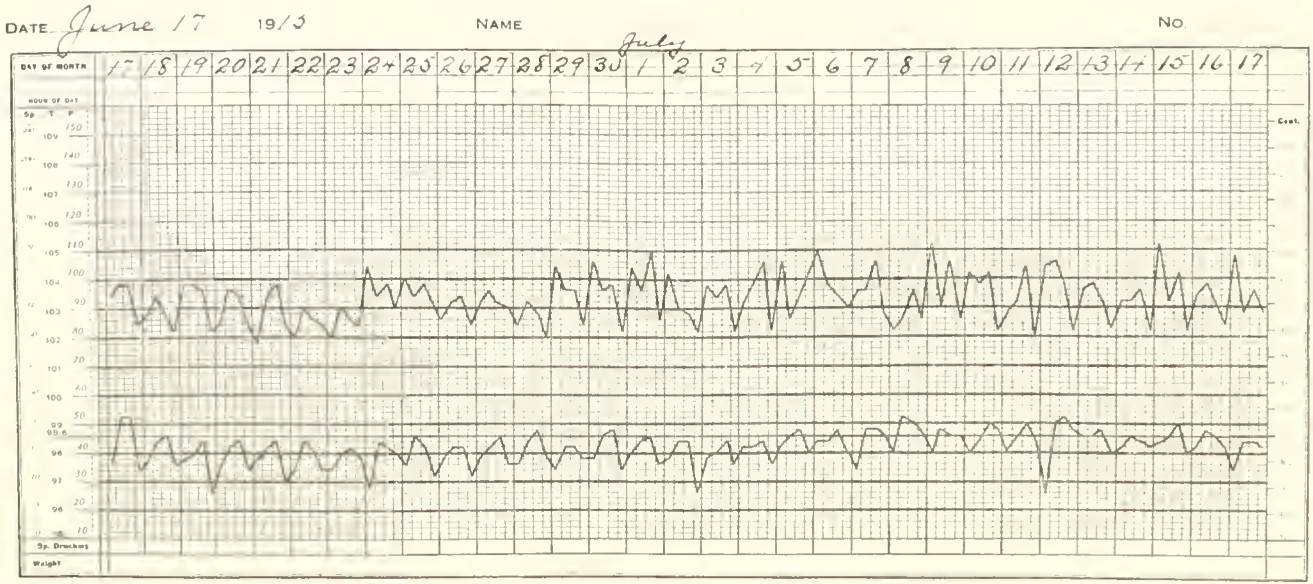


Fig 34.—Showing sympathetic irritation predominating. Pulse unusually high in relation to the temperature curve. Irregularities in pulse and temperature curves also indicate nervous instability.

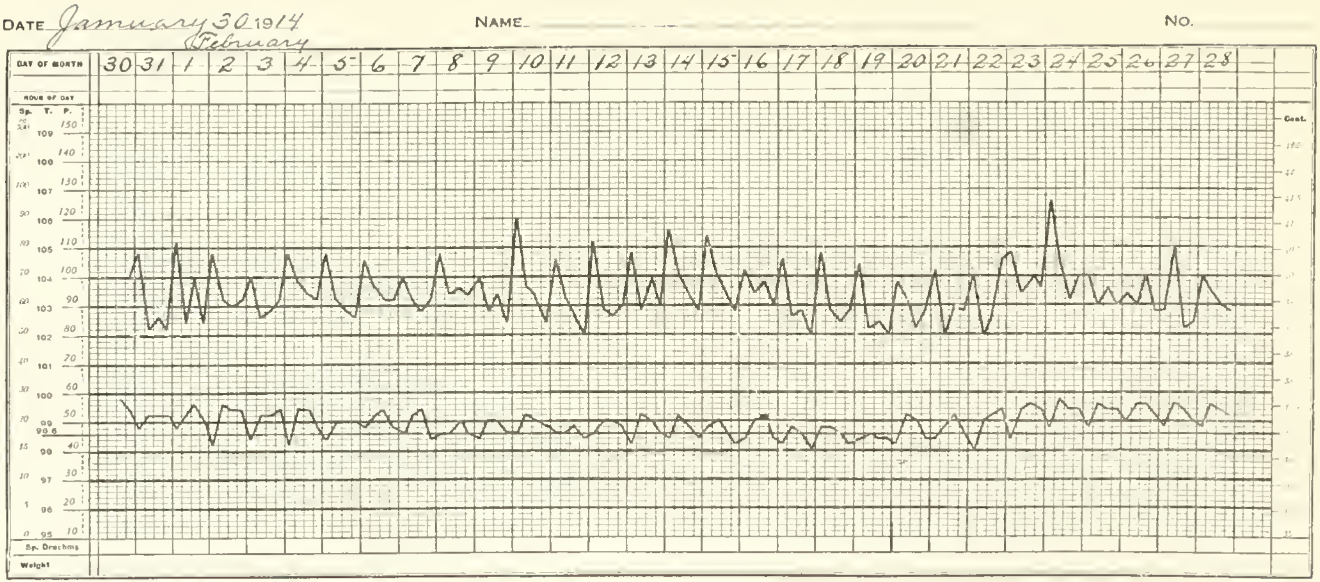


Fig. 35.—Illustrating a relatively high and irregular pulse curve in a patient in whom sympathetic tonus predominates. The degree of temperature as manifested in the chart is greater than would be expected from the degree of activity in the tuberculous process, and is most probably evidence of the nervous influence upon the temperature curve.

we must look upon the pulmonary involvement as being the determining factor in causing this low pulse. See pages 204 and 205.

Parasympathetic reflexes from other tissues will also cause inhibitory action upon the heart. Pressing upon the eyeball and irritating the nasal mucous membrane will both cause slowing of the heart.

Opposing this inhibitory action of the vagus is the accelerating action of the sympathetics, either through central stimulation by toxins; stimulation by the various emotional states which affect the patient; stimulation by certain internal secretions as the adrenal, pituitary and thyroid which result when the sympathetics are stimulated, or other factors which call for accelerated blood flow. Often the sympathetic irritation gains the upper hand and a markedly rapid pulse results.

Fig. 33 illustrates the manner in which the pulse as well as the temperature is influenced by toxemia. This chart is that of a patient who is suffering from moderately active tuberculosis. The course of the disease was interrupted at periods of from two to four or six weeks, with a high temperature. During each one of these rises in temperature there was an increase in pulse rate corresponding with that in the temperature curve. This is illustrated with the rise of temperature which started on the 6th of the month and culminated on the 9th. It will be noticed how the gradual increase in temperature and pulse rate followed each other. See page 207.

Figs 34 and 35 illustrate a marked disproportion between the degree of temperature present and the pulse rate; the pulse being very high considering the temperature curve. These patients were both nervous patients; and, while not inclined to worry, they were in a constant state of disturbed nervous equilibrium, which manifested itself upon the pulse. They both belong to the type of individuals with increased sympathetic tonus. See pages 208 and 209.

Figs. 36, *A* and *B*, are charts which illustrate sympathetic irritation in a patient who naturally is markedly vagotonic. This patient suffered from rather a widespread lesion in the right lung, which made satisfactory improvement. These are the early charts in the case. It will be seen in chart *A* that the temperature was ranging between 99° and 100°. The pulse during the first half of the month remained between 70 and 80, and dropped into the sixties. On the 14th of October this patient began to show marked signs of nervousness. She became exceedingly depressed, discontented, and unhappy. The effect is seen on the pulse. Without further elevation in temperature the pulse increased in rapidity, and, on several occasions, reached a maximum of 100°. This increased sympathetic stimulation occurred during the two weeks preceding the menstrual period and was probably induced by the action of the ovarian secretion upon the sympathetic nerves. It will be noticed that in chart *B* the pulse came down into the seventies; but, on the 8th of November, showed another elevation, which continued until the 21st. The sympathetic stimulation this time was not so marked. The difference was partly due to the fact that I had explained to this patient why she felt nervous and discontented, and, with this understanding, she was able to prevent a great deal of the depression which had manifested itself the previous month, and which had manifested itself more or less frequently throughout her menstrual life. See pages 212 and 213.

Intestinal Tract.—The antagonistic action of parasympathetics and sympathetics on the different portions of the intestinal tract is extremely interesting; and our understanding of this action will aid greatly in forming an accurate clinical conception of the digestive capabilities of the tuberculous patient.

Early in tuberculosis the toxemia present is not of a high degree, and its action upon the sympathetics is negligible, as compared with that present in the more advanced cases. From the very first, however, vagus stimulation seems to be important. This is shown in the larynx, in hoarseness due to interference with the innervation; in the irritation which produces cough, and in the increased bronchial secretion. It is likewise marked in the intestinal tract as I shall now proceed to describe.

Influence on the Salivary Flow.—During tuberculosis, sometimes there is an increased amount of saliva and, at other times, there is a decrease. These changes show more particularly in some patients than in others. There is a double secretory nerve supply to all the buccal secretory glands, the parotid, submaxillary, sublingual, and retrolingual glands. Stimulation of either

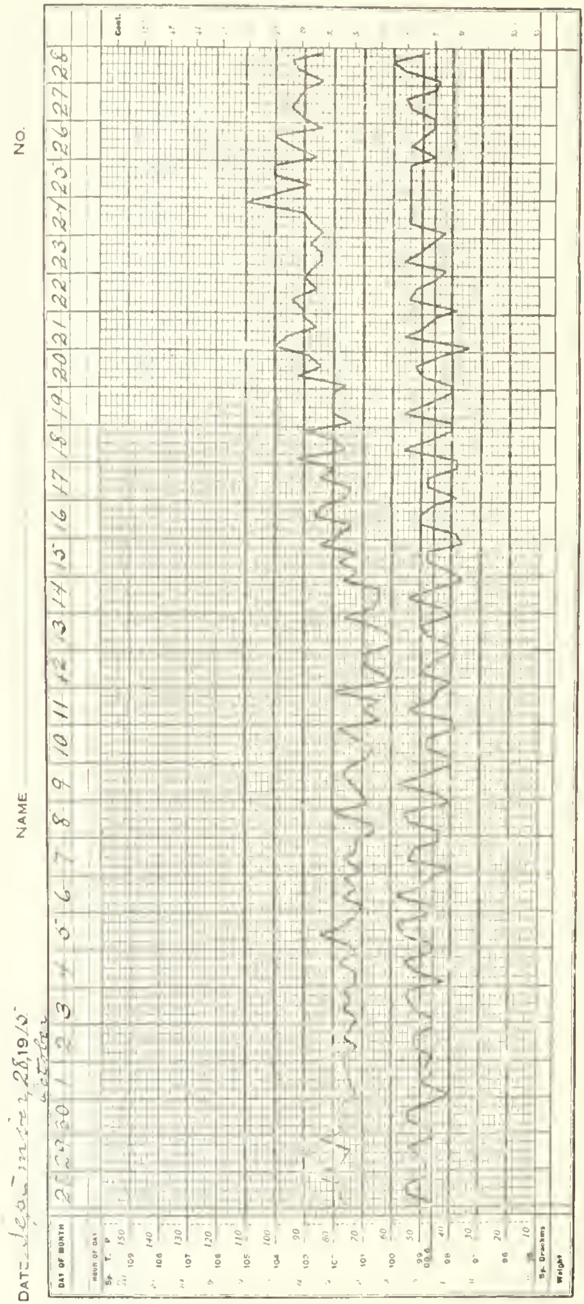


Fig. 36*A*.—Illustrating sympathetic irritation due to the stimulation of the ovarian secretion during the two weeks prior to menstruation. The degree of rapidity of the pulse was accentuated by anxiety, worry, and discontent.

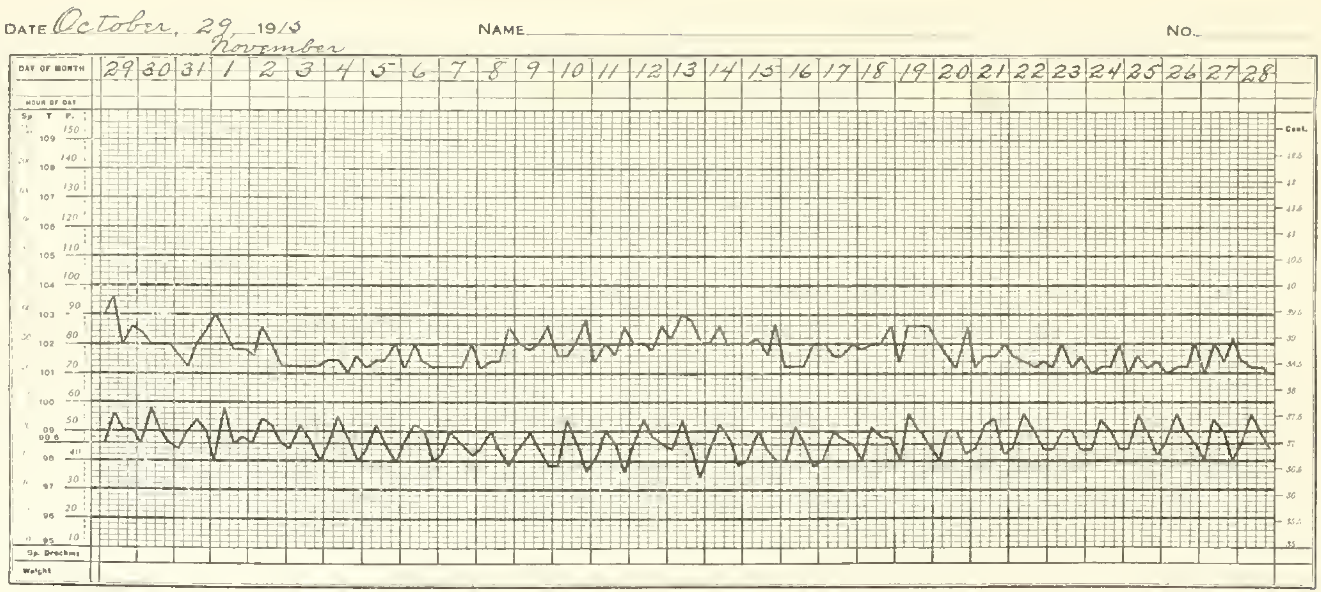


Fig. 36B.—Illustrating slight elevation of the pulse from the 8th to the 21st of November, the two weeks prior to menstruation, but much less severe than at the previous period; relief being due to freedom from the severe nervous irritation noted in Fig. 36A.

the chorda tympani, or of the superior cervical ganglion will decrease the salivary secretion. It would seem that, clinically, the parasympathetics must be the more important secretory nerves because the toxemia which stimulates the sympathetics, also the atropin which inhibits the action of the parasympathetics produces dryness of the mouth.

Tongue Atrophy.—As a result of prolonged chronic inflammation in the lung the tongue atrophies and when protruded it may push toward the side of the more destructive lesion. This reflex is produced by afferent impulses coursing in the vagus and efferent through the hypoglossal.

Atrophy of Facial Muscles.—The muscles of the face on the side of a markedly destructive lesion often atrophy, the path of the afferent impulse being through the vagus, the efferent through the trigeminus.

Motor and Sensory Disturbances in Pharyngeal Structures.—Patients suffering from chronic pulmonary tuberculosis suffer from reflexes which affect the pharyngeal tissues. Parasympathetics supply the mucous membrane with sensation and the mucous glands with secretory power. These fibers when reflexly disturbed may show either sensory or motor phenomena. The increased sensitiveness of the pharynx which is particularly noted when a pulmonary process is acute, as during necrosis and cavity formation may be accounted for in this way. During this time cough is frequently followed by retching and vomiting. Slight irritation of the pharynx, such as is caused by accumulation of mucus, may cause vomiting. The ease with which the vomiting reflex may be exerted by tickling the pharynx is well known; and when hypersensitive, the reflex phenomena follow the more readily.

A motor reflex in the pharyngeal muscles is often noted late in the disease. The pharyngeal muscles also atrophy the same as other structures whose central cell bodies are in reflex connection with chronic constant stimulation. Thus, the irritated conditions of the nerves of the pharynx may show as vague pains or as an atrophy of the muscles, or as an occasional disturbance in the act of swallowing.

Coated Tongue.—A dry coating of the tongue is often noticed during periods of temperature in cases where toxemia is a factor.

The drying effect on the salivary secretions through the sympathetics must be thought of as an etiological factor. The fact that this is nearly always accompanied by a diminution of appetite and deficiency in gastric and intestinal efficiency makes the cause the more probable, for these other conditions are likewise due to deficient parasympathetic, or increased sympathetic, tonus. When toxemia has passed over, on the other hand, a normal tongue, and even increased appetite, may be seen.

Stomach.—The disturbances on the part of the stomach in early tuberculosis are those which belong to the class of so-called nervous dyspepsias. The form which seems most common, except when the patient is suffering from toxemia or mental depression, as I shall describe later, is hyperactivity. This is caused reflexly through the vagus, the reflex coming from the inflamed pulmonary parenchyma. At first the patient's digestive powers are, if anything, above par. This is one reason why the tuberculous patient is able to care for such large amounts of food. Associated with increased secretion there is often an increased motility. This shows itself now and then in a feeling of nausea and tendency to vomit. These conditions, however, are not continuous. The increased vagus tonus can be relieved by the administration of atropin; and, following its administration, hyperacidity and hypermotility often disappear or are ameliorated.

There are also several factors which come in to cause sympathetic stimulation and inhibition of the vagus action, noticeably, toxemia and the depressed nervous states which are so common and which are characterized by mental depression, discouragement, pain and fear. These emotional states come and go through the disease. They are sometimes dependent upon and sometimes independent of toxemia.

Intestines.—A similar condition obtains in the intestinal tract where we have states of increased parasympathetic tonus, causing increased secretion and increased motility, and abnormal sympathetic irritation inhibiting this action and interfering with secretion and peristaltic action. Spastic constipation as a result of increased parasympathetic tonus is common in early tuberculosis, while the atonic type is the rule later.

Definite effects of each system are not so easy to point out in the intestinal tract because of the preponderance of stasis and constipation, which are regularly found in general life and which affect so many of those who are afflicted with tuberculosis prior to the time when the disease becomes manifest. We may say, however, that, as a rule, digestion becomes more impaired and stasis and constipation become more pronounced as the disease advances and toxemia and the various depressive emotional states become more marked. Thus it would seem that early tuberculosis is a condition in which parasympathetic tonus naturally predominates over sympathetic stimulation; and advanced tuberculosis a condition in which now the tonus in one system, now in the other predominates. The degenerations which occur after the disease has existed for a long time are accountable for many of the symptoms.

In offering this analysis of nervous action in tuberculosis, I realize fully that it is impossible to always point out the direct relationship between cause and effect; because we are dealing with a disease which produces dysfunction on the part of many organs and which results in a multitude of conditions which might produce symptoms similar to those which could be explained at one time by irritation of the sympathetics, at another by irritation of the vagus. It can be seen, however, that these two systems are to be taken into account; and that, as long as inflammation in the lung exists, so long are impulses carried to various organs through these two systems, which, acting antagonistically, disturb the normal equilibrium, which is so necessary to normal function.

CHAPTER VIII.

THE NERVOUS SYSTEM CONTINUED: THE RELATIONSHIP OF THE SYMPATHETIC NERVOUS SYSTEM TO TOXEMIA AND THE DEPRESSIVE EMOTIONAL STATES IN TUBERCULOSIS.

The clinician must ever turn to physiology and pathological physiology for the explanation of phenomena which he observes in medicine. The study of medicine, however, increases greatly in interest as he is able to assign a plausible reason for observed facts. Volumes have been written in explanation of the phenomena of anaphylaxis. Temperature has been discussed in every text book dealing with diseases accompanied by toxic conditions. The symptoms which accompany toxic states are well known, and more or less accurately described, yet a fully satisfactory explanation of the rationale of their production has not been offered.

Primarily I believe that it is justifiable to state that the symptoms which accompany a disease are at least partly associated with the means which the organism provides for its defense against that disease. It may be difficult at times to see how this works out, especially when the symptoms become so serious as they often do when the organism succumbs to the disease. Our answer to this is that it is not the symptoms which cause death but the disease itself; and that the disease may become so serious that the very means which have been developed for the defense of the organism may become abnormal and even harmful.

To understand this better I would cite instances of physical defense in which the means of defense proved harmful. At the time of the Lusitania disaster the story is told of a woman and three men who seized hold of a piece of wood which was sufficient to help them all to keep afloat. Nature turned all the latent forces within their bodies to active working power and gave them almost superhuman strength of muscle to preserve them from death; but, in spite of this, one by one they let loose their hold; and all perished but one. The unnatural strength which was thus developed, and which would have saved everyone of them, had the struggle not been too long, ended in exhaustion, and permitted the enemy against which they were struggling, to overcome all but one; and she was nervously and physically exhausted for days, as a result of the struggle. Many instances are also cited in the present European war where men have been compelled to defend themselves by supreme effort. The retreat from the Mons to the Marne is a case in point. Physical and nervous energy were developed in the troops who made that retreat to an extent that seems incredible. For nine days men marched, averaging twenty miles a day, harassed continually by an enemy. They were enabled to do this because the necessity of self-preservation demanded it and the latent powers within them were called into action and every particle of force which the body could muster was converted into muscular energy to be utilized in making their escape. This supreme effort was followed by exhaustion. Many men fell by the wayside, and those who succeeded in making their escape were exhausted nervously and physically. Many of them sank into a sleep which lasted two or three days; some to waken no more. The exhaustion was a result of the overworking of the means of defense, particularly the sympathetics, adrenals and thyroid.

The same is true in infectious diseases. The body is confronted by an enemy. Its defensive powers are called out, not for the supreme physical effort only, but for a chemical effort, requiring energy none the less. Bacteria liberate foreign protein molecules when they go into solution and during their growth. These molecules are set free in the tissues. As a result of chemical action they are destroyed, liberating toxic molecules. If these toxic molecules are set free in great numbers the organism may be unable to stand their action and death may result. When infectious microorganisms enter the body, if implantation results, the metabolic processes of the body are increased. As a result of this increased metabolism, there is an increase in heat production. The toxic molecule, which results from splitting up the protein molecule, is set free and acts upon the cells of the central nervous system. It stimulates the sympathetics, adrenals and thyroid, and, through its action upon them, produces a constriction of the superficial vessels, interfering with the liberation of heat. As the combined result of the increased oxidation and decreased elimination of heat, the body temperature rises. This increase in temperature has a tendency to check further multiplication and growth of bacteria. Whether or not this is a part of the program of defense, it is difficult to say; but an increase in temperature, particularly when it reaches a maximum of 100° or 101°, or more, cannot help exerting an inhibitory influence on the growth of bacteria.

Whenever the bodily forces are called upon for defense, whether it be to attack an enemy, to escape from an enemy, or to fight an infection, the processes which go on within the body are the same in principle. Latent energy is transformed into active working energy; and, in order that the protective forces of the organism may be as great as possible where the danger is serious, the enteral viscera are called from duty for the time being, their function being temporarily inhibited that the energy ordinarily used up by them may be devoted to defense.

Crile[1] has shown that the particular organs which are concerned in defense are the brain, the adrenal gland, the thyroid gland, the liver, and the muscles. These, he speaks of as the "Kinetic system," a system which is "evolved primarily for the transformation of latent energy into motion and heat;" the motion and heat being the two forms of energy which are utilized by the organism in defense. These organs are all more or less interdependent in their action. Cannon[2] has especially studied the action of the adrenal glands and shown how, owing to the fact that it is innervated through the sympathetics (splanchnics) its action is dependent upon the same stimuli as those which affect the sympathetics in general. He has also shown that the effect of adrenin which results from this stimulation when thrown into the circulation acting peripherally upon the myoneural junction of the sympathetics is to further produce or prolong the same action as results from central sympathetic stimulation. Thus, adrenin in the blood causes dilatation of the pupil, rapid heart action, a checking of the gastrointestinal secretions and motility, stimulation of the thyroid and a setting free of glycogen from the liver, phenomena, all of which represent sympathetic stimulation.

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[1] The Origin and Nature of the Emotions, W. B. Saunders Company, 1915; Man—An Adaptive Mechanism, The Macmillan Co., New York, 1916.

[2] Bodily Changes in Pain, Hunger, Fear, and Rage, D. Appleton & Co., 1911.

The central nervous system is the constant recipient of impulses. These impulses, if sufficiently strong, call forth a response which results in action. Every conceivable agency acting upon the body, whether it be a toxin, a protein, pain, joy, sorrow, discontent, the pleasure of a beautiful landscape, or the horror of the battlefield, produces impressions upon the central nerve cells which, when sufficiently strong, produce action in accordance with the type of the stimulating agency. Impulses of lesser degree are received without causing action. One peculiar characteristic of stimulating agencies is their selective action upon peripheral nerves. One impulse may act through somatic nerves, another through sympathetics, and still another through parasympathetics. There are many instances in which the action is even more selective than this; where only one portion of a system, for example, or a single brain center, is stimulated.

Knowing the selective action of the digestive juices, and the selective power of body cells to take from the blood stream the particular substances required for their growth and function, and to reject those which are valueless or harmful, and appreciating the selective action of the substances secreted by the various glands of the body, we are prepared to believe that the purposes of the organism are best served by a nervous mechanism which is also selective in its reaction to harmful stimuli.

We observe that certain stimuli in which we are led to believe that primary action is peripheral, act principally upon the nasal parasympathetic nerves, others upon the bronchial, others upon the cardiac, and still others upon the gastrointestinal branches. Other central stimuli act upon the sympathetic and still others upon either the sensory or motor nerves supplying the skeletal structures.

It may be that this should be spoken of as a selectivity in response or reaction rather than, in action; for, it may be that the impulses which meet the nerve cells affect all, but only produce response or reaction in definite groups. This latter suggestion seems to hold for those groups of bodies which produce toxemia and anaphylaxis. The syndrome of toxemia is central stimulation plus the syndrome produced by peripheral activity in sympathetic structures; while the syndrome of anaphylaxis is that of central stimulation plus the parasympathetic syndrome caused by peripheral irritation of the nerve endings of the particular branches of the parasympathetics which show the irritation.

Toxemia, worry, fear, pain, etc., produce certain reactions on the part of the body which have the effect of neutralizing their own harmful influences. A long continued action of these harmful stimuli causes overwork and overstimulation, which leads to a degree of exhaustion of the protective mechanism. This leads to neurasthenia, exhaustion or perversion of the action of the nervous system; psychasthenia, exhaustion or perversion of action of the higher or psychical centers; myasthenia, exhaustion or perversion of action of the motor mechanism; in fact, we might express this whole condition as a general cellular and psychical exhaustion or perversion of action.

As a clinical picture we see this in all degrees of severity. We see it as an acute process in such conditions as acute toxemia and shock, and as a chronic process in such states as the protracted toxemias, and the prolonged nervous depressions. Perverted action on the part of the important organs of the body under such circumstances is known to us all. A study of the changes in the important organs as a result of these harmful stimuli has been made by Crile[3] and his associates by which they have been able to show the effect of harmful stimuli upon the brain, adrenals, thyroid, and liver. Crile thus approaches the subject of the effect of harmful stimuli (among which are diseased conditions) upon the body by studying the impression that these stimuli make upon the mechanism of defense. His work shows that changes in the brain, adrenals, thyroid, and liver follow either marked harmful stimulation of short duration, or less severe stimulation when prolonged.

With these suggestions before us we are now prepared to study the syndrome of toxemia. We must look upon the symptoms which accompany toxemia as being a result of the attempt of the body to destroy the toxins, as well as a result of the action of the toxins themselves.

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[3] Man—An Adaptive Mechanism, Macmillan, New York, 1916.

When bacteria, or any other foreign protein enters the circulation, it is split up into toxic and sensitizing molecules (Vaughan). The toxic molecules act upon the cells of the central nervous system and cause them to send stimuli peripheralward, particularly through the sympathetic system to the tissues supplied by it. The effect through the sympathetics is widespread. At some period during the toxic state, depending upon the degree of toxemia present, there is a stimulation of practically all the tissues and organs supplied by the sympathetic nervous system. This is noted in the epidermal tissues as: first, a stimulation of the pilomotor muscles, causing the condition of "goose flesh" which very often appears preceding or during the chilly sensations which result from toxemia; and, second, in the stimulation of the muscles to the sweat glands, causing an increase of perspiration.

The effect upon the vasomotor system differs with the strength of the stimulus. There is a stimulation of the sympathetic fibers exerting an inhibitory action upon the internal organs which are activated by the parasympathetic nerves, as shown by the lessening of the gastrointestinal secretions, and by the reduction in the gastrointestinal motility. The same sympathetic stimulation increases thyroid activity and hastens the pulse rate. Stimulation of the adrenal glands is shown by the increase of secretion of adrenin, which is accompanied by a forcing of glycogen from the cells of the liver.

The effect of this stimulation is a general inhibition of function upon all the tissues supplied by the parasympathetic system. The purpose of the organism, for the time being, is defense; consequently, the functions of organs which are not immediately necessary for that purpose, are checked. The demand for energy required by the digestive system is temporarily removed and the needs of the body are supplied by other methods. This is particularly noted in the fact that the glycogen stored up in the liver is brought out into the circulation for consumption. The adrenal glands are stimulated, and they, in turn, stimulate the entire mechanism of defense, bringing the brain, the liver, and the thyroid gland into action in the transformation of latent energy into active defensive forces.

As a result of this sympathetic action, there results a group of phenomena, which make up the syndrome of toxemia. This is the same, except in minor details, no matter from what source the toxemia comes. The syndrome varies according to the degree of toxemia present, and whether the inoculation of toxin is single or multiple. Among the most common symptoms belonging to this syndrome are malaise, lack of endurance, nervous instability, loss of strength, rapid heart's action, lack of appetite, furred tongue, and hyposecretion and hypomotility throughout the gastrointestinal tract. It can be seen that this group is definitely the picture of central stimulation plus an expression peripherally of stimulation of the sympathetic system.

While a temporary inhibition of function may be permitted, and the energy conserved may be utilized in defense, if long continued, it appears to be deleterious; consequently, this sympathetic stimulation should be relieved as quickly as possible that the vegetative functions may go on undisturbed; for, while a temporary disturbance of these functions might or not be serious to the individual, a prolonged disturbance produces injurious results.

The sympathetics are irritated under several different circumstances in tuberculosis, as follows:

1. Whenever tubercle bacilli are multiplying or going into solution,—whenever they are producing and liberating toxins.

2. Whenever, as a result of the process, tissues break down resulting in protein absorption.

3. Whenever other bacteria complicate the process and give off their toxins into the circulation.

4. When suffering from depressive emotional states such as those induced by disappointment, discouragement, dissatisfaction and fear or worry over domestic or business affairs, sometimes, the sympathetics, at other times, the parasympathetics dominate the picture.

From this it can be seen that toxemias and depressive emotional states as they occur in tuberculosis act centrally and at the same time cause peripheral sympathetic action; and are productive of inhibition of function of many internal viscera, and an elevation of temperature and general loss of nervous and physical force. The picture of general neurasthenia manifests itself with its irritability of nerve centers and general loss of physiological balance whenever the process is long continued.

Toxemia is an active force in tuberculosis whenever the tuberculous process is active. It is most marked when the process is most acute. The system gradually grows accustomed to the presence of toxins and adjusts itself by the formation of a tolerance or defense. When this state has been attained a considerable amount of toxins may find its way into the circulation without producing marked symptoms. In fact, after the period of acute activity has passed, it is the rule for the so-called toxic symptoms to disappear. They can be brought on again, however, by exertion. Exertion could act either by forcing more toxins out into the circulation as is generally stated (so-called autoinoculation) or by oxidizing the toxins more rapidly as a result of the increased oxidization attendant upon the extra work produced. The tuberculous patient who rests quietly is free from the symptoms of toxemia as a rule, except during the periods of growth and destruction of bacilli and extension of the process and during the time of active breaking down of tissue. Toxic symptoms are increased by exertion and diminished by rest; consequently rest is an important factor in treatment during the toxic state.

The more persistent symptoms belonging to the toxic group are those which result from various degrees of exhaustion of the machinery of defense including both neurons and endocrine systems; malaise, lack of endurance, loss of strength, and nervous instability. These persist much longer, as a rule, than the symptoms on the part of the internal viscera such as rapidity of heart action, diminution of secretion and diminution of motility of the gastrointestinal tract. The organs of the body seem to have a wide range within which they can functionate fairly normally; and, they seem to be able to withstand considerable abuse and still carry on their part in the animal economy.

The same syndrome may be present during depressive emotional states as during the states of toxemia, although the symptoms are, as a rule, milder in character. Thus, the patient who is in pain, also the one who is disappointed, fearful, anxious, discouraged, pessimistic and homesick, likewise the one who is discontented, cross, irritable and angry, suffers from a degree of nervous exhaustion and general inhibition of functions of important viscera. These conditions depress the functions of the internal viscera and lower the power of fighting the disease. Therefore, pain should be relieved if possible and the patient should be kept as cheerful and happy as possible. In a recent paper[4] I discussed the relationship of the group of symptoms which accompanies toxic conditions to the sympathetic nervous system, and, while I made the discussion general, it applies to the subject under discussion. I quote the paper extensively, although by doing so, a slight amount of repetition is unavoidable.

"The human body under normal conditions has a perfect physiological balance. The relationship of one part or organ is in a state of perfect balance with every other part or organ. A disarrangement of this equilibrium produces pathological conditions and dysfunction. The derangements are indicated by symptoms and signs, which, when grouped, give us a picture of definite disease.

"The explanation of well known symptoms is often wanting, the practitioner being compelled to learn them as a part of the symptom-complex, belonging to a certain disease instead of being able to point out the exact forces operating to produce them. An etiological classification of symptoms leads us to a better understanding of the disease in question and facilitates diagnosis.

"In analyzing the symptoms in early pulmonary tuberculosis, I found that they could all be classed etiologically in three groups: 1, those due to toxemia; 2, those due to reflex action; and, 3, those due to the tuberculosis process *per se*, as shown in the table on page 226.

"In this manner we classify some twenty-six symptoms which belong to tuberculosis in three groups so that each symptom in the individual group is due to a common etiological factor. Such a classification simplifies diagnosis. It offers an explanation for the fact which has long been observed,—that symptoms are variable in that the same ones are not always present, and when present, they are not always prominent.

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[4] The Syndrome of Toxemia, an Expression of General Nervous Discharge Through the Sympathetic System, Journal American Medical Assn., Jan. 8, 1916, vol. lxvi, pp. 84 and 85.

"When studying these three groups of symptoms I was impressed with the fact that there was nothing distinctive of tuberculosis in that group which is due to toxemia. The same symptoms could be due, as well, to an infection of the tonsil, the prostate, the fallopian tube, a toxemia from intestinal stasis or an acute infectious disease. They are, in short, a part of the syndrome of infections in general. I was further impressed with the fact that the second group, those of reflex origin, all point to organs other than the lung, but that they belong to organs which are supplied by the parasympathetics and sympathetics, both of which supply the lung. *A further analysis of the group which is due to toxemia shows that these symptoms are due to an action upon the central nervous system, also a peripheral effect upon the sympathetic structures and the glands of internal secretion.*

GROUP I.	GROUP II.	GROUP III.
TOXEMIA *	REFLEX ACTION.	TUBERCULOUS INVOLVEMENT PER SE.
Malaise	Hoarseness	
Lack of endurance	Tickling in larynx	Frequent and protracted colds
Loss of strength	Cough	
Nervous instability	Circulatory disturbances	Spitting of blood
Lack of appetite	Digestive disturbances	Pleurisy
Digestive disturbances	Loss of weight	Sputum
Loss of weight	Chest and shoulder pains	
Rapid pulse	Flushing of face	
Night sweats	Apparent anemia	
Temperature		
Anemia		

"To make this clearer it is necessary to recall that the vegetative nervous system supplies impulses to structures which are not controlled by the will. These are the organs supplied by smooth muscle such as the stomach, intestines, blood vessels, ducts of glands; also certain organs possessing striated muscle fibers such as the heart, the beginning and terminal portions of the alimentary canal, and the generative organs.

"The vegetative system is divided into three groups; 1, the cranial and bulbar, which are spoken of as the parasympathetic system; 2, the thoracic and upper lumbar, known as the sympathetic system; and 3, the lower lumbar and sacral, known as the sacral system.''

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*Since writing this paper, I have been able to explain several other symptoms as belonging to this group, such as headache and general aching; and, when the toxemia is very severe, producing collapse, there results: 1, vasodilatation (vasomotor paralysis); 2, sweating; 3, subnormal temperature.

The parasympathetic system activates head and enteral structures: It contracts the pupil, increases salivary secretion, slows the heart beat, causes an increase of gastric and intestinal juices, and furnishes motor power for the gastrointestinal tract down to the ileocecal valve. The sacral parasympathetic fibers supply the lower bowel from the ileocecal valve of the anus, the bladder and certain of the generative organs. The sympathetic system sends branches to most organs supplied by the parasympathetic system; and where the fibers from these different systems meet there is an antagonistic action between the sympathetics and the parasympathetics. If the sympathetic stimulation is sufficiently strong, it overcomes the parasympathetics. If this control is only momentary or of short duration, as it is in fear and anger, as shown by Cannon[5] the general strength of the individual is increased. He possesses for the time being, a power greater than normal. On the other hand, if this sympathetic control continues, a general inhibition of function in the organs activated by the parasympathetics takes place and the individual's powers suffer a diminution; thus, the dry mouth, lack of desire for food, stoppage of digestion, and rapid heart's action, which are temporary in the presence of the major emotions, are emphasized and prolonged during toxemia and are expressed as a general malaise, a more or less continuous absence of appetite, coated tongue, retarded digestion, constipation, rapid heart action and tendency to perspiration.

While impulses may be carried directly from the brain and cord to the skeletal muscles, insuring immediate and selective response and while the parasympathetic connector fibers run directly from the central nerve cells to the peripheral cells in the organ, thus giving a limited response, the sympathetic connector fibers as a rule divide and pass to many ganglia supplying various organs. These ganglia act as distributing stations for sympathetic impulses and make their action general, which accounts for the fact that so many organs and parts are involved in sympathetic irritation.

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[5] Bodily Changes in Pain, Hunger, Fear, and Rage. D. Appleton & Co., 1915.

That the effect of toxemia is not wholly expressed through the nervous system is now certain. While the peripheral effect is that of sympathetic action, yet we must bear in mind that this may be partly due, or, at least, prolonged by the internal secretion of the adrenals which is increased by the same stimulation as that which produces the general sympathetic stimulation. Cannon has shown that the emotional states such as anger and fear, while being an expression of general sympathetic discharge, may be kept up by adrenin in the blood. The adrenal glands are supplied through the splanchnics, and, impulses which cause a general sympathetic stimulation, stimulate these glands also. A minute amount of adrenin poured into the blood stream has the effect of producing a prolongation of the condition which is brought about by direct sympathetic stimulation; thus adrenin will cause a dry mouth, impaired digestion, intestinal stasis and a rapid heart. That toxemia, like the emotional states, acts by stimulating the sympathetics and by prolonging the action through the stimulation of the adrenals seems quite certain. It is also known that disturbances in the thyroid and pituitary, may also have a part in this general picture; but even this does not alter the fact that the syndrome of toxemia is an expression of general sympathetic discharge.

Further studies show me that my early papers do not emphasize sufficiently strongly the fact that toxins and depressive emotional states act centrally, causing a general irritability of the central cells. The peripheral expression of inhibition through the sympathetics, however, is well emphasized.

Physiologists tell us that the action of adrenin upon the internal viscera is produced through peripheral stimulation instead of central stimulation of the sympathetics. The particular point in the reflex arc which is acted upon is the myoneural junction where the nerve endings and the muscle cells join. P. J. Meltzer and Clara Meltzer[6] proved that adrenin acts peripherally instead of centrally by studying its action upon the vasoconstrictors in a rabbit's ear. The vasoconstrictors to the rabbit's ear pass through the sympathetics from the superior cervical ganglion, and through

[6] The Share of the Central Vasomotor Innervation in the Vasoconstriction Caused by Intravenous Injection of Suprarenal Extract, American Journal of Physiology, vol. ix, p. 147.

the auricular magnus, a branch of the third cervical nerve. By cutting the third cervical nerve and removing the superior cervical ganglion, connection with the central nervous system was destroyed. Regardless of this fact the intravenous injection of adrenin still produced a constriction of the vessels of the rabbit's ear, showing that the action must be peripheral instead of central.

CHAPTER IX.

THE CIRCULATORY SYSTEM IN TUBERCULOSIS.

A good heart is the best asset that a patient suffering from tuberculosis can have. It serves him well during the time when he is struggling with the disease as an infection capable of arrestment; and it usually proves to be the determining factor as the disease progresses.

The question of the displacements of the heart as they occur in advanced tuberculosis will be discussed fully in Chapter XI so it need not be repeated here.

The heart begins to feel the effect of tuberculosis as soon as the disease is of sufficient magnitude to cause an appreciable diffusion of toxins into the tissues or to interfere with inspiration by producing changes in the elasticity of the pulmonary tissue or by reflexly disturbing the muscles of respiration, or reflexly influencing the heart itself through its nervous mechanism.

Nervous Influences upon the Heart in Tuberculosis.—It is impossible to understand the symptoms on the part of the heart in tuberculosis unless we have a definite understanding of its nerve control and the factors which are present to influence this.

In discussions upon the heart in its relationship to this disease it is usually said that a rapid pulse is one of the early signs. It is also considered to be a factor throughout the entire disease. This statement, however, must be modified, as can be readily seen by observing tuberculous patients both at rest and during exertion, during the various phases of the disease.

The pulse rate in tuberculosis is variable because the conditions which affect the nerves that innervate the heart are variable and the nerve stability itself is variable.

In order to understand the influences which act in increasing and decreasing the rate of the heart beat it is necessary to bear in mind that the heart is supplied by both the sympathetic and vagus divisions of the vegetative nervous system. The sympathetic has a tendency to accelerate and the vagus to slow the heart action. Toxemia acts by stimulating the sympathetic nervous system centrally, consequently toxemia manifests itself on the part of the heart by increased rapidity of contraction. As a result of pulmonary tuberculosis we must bear in mind also that the nerve endings of both the sympathetics and parasympathetics are irritated by the inflammation in the lung and that the latter shows reflex action in the heart and other visceral structures. On the part of the heart, reflex stimulation of the vagus tends to decrease the rapidity of contraction; consequently, we have the heart subject to central stimulation of the sympathetics by the toxins, and peripheral stimulation of the vagus by the inflammation in the lung, the former tending to increase, the latter to slow, the contractions. As a result of this action, as a rule, the parasympathetics are overcome for the time and the heart shows increased rapidity. When the toxemia has passed over, however, the central stimulation of the sympathetics ceases. Then the heart is subject only to the reflex slowing influence through the vagus from the inflammation in the lung. It must be understood that I am now speaking only of direct nerve influence. There are many other factors which come in and will be discussed in the proper place.

In many patients the vagus and sympathetic stimulation balance each other, the normal equilibrium is maintained, and the normal number of heart contractions result. In other cases the vagus predominates over the sympathetic and we find the pulse even slower than normal. In still others, however, the sympathetic action is the stronger and the pulse remains rapid.

It has long been said that a persistently rapid pulse in tuberculosis means a bad prognosis. While this is not invariably true, there is some ground for it because it means that the patient's sympathetic tonus is in the ascendency; and this, being the inhibitory system, causes a diminution in the gastrointestinal functions and in the functions of all the other important viscera of the body. It would be more rational to state that those patients who are distinctly of the sympathicotonic type, or who have a decreased parasympathetic tonus, which means practically the same thing, are not as able to withstand the disease as those who have increased parasympathetic tonus. Charts shown in Chapter VII, Figs. 29-36 inclusive, illustrate the peculiarities of the pulse here described. The particular characteristics of the pulse in early tuberculosis, which are dependent upon disturbances in the heart's nervous mechanism, are: first, rapidity during periods of toxemia; second, either a normal, an increased or a decreased rapidity during periods of quiescence, while the patient is at rest; third, instability of the pulse on exertion; its reaching a higher point and returning to the normal slower than usual, being a very common condition. (Figs. 29-36, pages 200-213.)

After acute toxemia has passed over, toxic symptoms may continue through faulty methods of living and continue to show in acceleration of the pulse rate. Such emotions as disappointment, fear, anxiety, discouragement, as described in Chapter VIII, have the effect of producing and prolonging the same symptom-complex as that produced by toxemia, though, as a rule, the symptoms are not severe. Consequently, if the patient is discouraged and disappointed these conditions stimulate the sympathetic nervous system and produce rapid heart action. The accompanying chart (Fig. 37) illustrates this well. This patient was on duty as a nurse in the hospital. She was suffering from tuberculosis and was impressed with the fact that it meant death. After examination I gave her hope and told her that she could get well, and firmly impressed her with the idea that she would be restored to health. She continued her work just the same, but the effect of relieving her of fear and its depressing action upon the sympathetic nervous system is well shown in the appended chart.

The influence of the nervous system upon the blood vessels can be well understood by the fact that the sympathetics supply the muscular coats of the blood vessels of not only the internal viscera, but of the skeleton as well with all nerve fibers. Consequently, anything that disturbs the sympathetic nervous system is apt to find expression in the changes in vascular tone.

There are other changes to be considered in the production of unstable heart action. It is impossible to determine how much of the effect is produced by one cause and how much by another. The problems become more and more intricate as the disease advances and increases in complexity. At first, the general tissues of the body are disturbed little, if any. At this time the heart muscle is sound, unless injured by other factors. What-

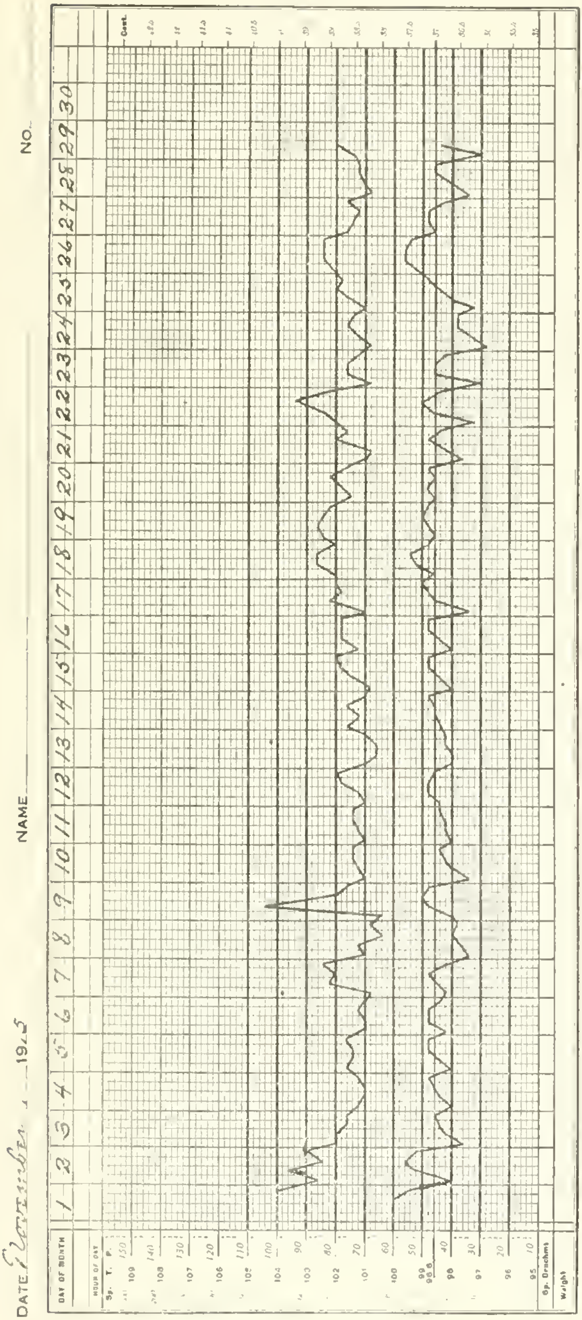

Fig. 37.—Illustrating increase in temperature curve and high pulse rate due to worry and fear on the part of the patient that she might have tuberculosis, believing that the same meant death. The temperature was relieved from the maximum of 100 degrees to practically normal within three days after the patient was examined and given hope. The marked rise in the pulse on the 9th was due to worry over trying to make arrangements for treatment. The rise in temperature and pulse from the 15th to the 23rd was premenstrual. The rise on the 26th was due to overwork in getting ready to give up her position. Each rise in temperature was due to factors which stimulated the sympathetics.

ever changes are present, as a result of the tuberculous process, are due to the central stimulation of the sympathetic and endocrine systems by the toxins; the peripheral stimulation of the vagus system by the inflammation in the lung; and to the disturbance of the inspiratory act, interfering with its accessory aid to the circulation. Later, however, these causes are exaggerated and to them are added the factors of malnutrition and general wasting; general degenerative changes as well as those affecting the heart muscle and blood vessels; displacement of the heart; further disturbances in internal secretions; and the destruction and other changes in the pulmonary tissue.

Physiological Facts.—To understand fully the effect of these deleterious factors on the heart and circulation we must have accurate physiological conceptions. The circulation of the blood is for the purpose of adding oxygen and combustible materials to the tissues and removing waste. This interchange takes place in the capillaries. It is necessary that the flow of blood through the capillaries be rapid that this exchange may be carried on properly. It is further necessary that the blood be conveyed to them with adequate force and be carried away with proper dispatch. The blood pressure of the systemic arteries depends upon the amount of blood delivered in systole and the tension in the systemic vessels. It will be understood from the discussion in this chapter that the output at each systole is decreased and that the muscular tone of both heart and vessels is altered, particularly in the advanced disease.

The lesser circulation varies somewhat from the systemic. The blood pressure here depends on the same factors as in the greater circulation, but the tissues are so different in texture, and the conditions so different, that the pressure is much less. The loose spongy pulmonary tissue offers little support in comparison with that offered by the solid organs and firm skeletal muscles. So does the negative intrathoracic pressure offer a reduced resistance to the vessels within the thoracic cage as compared with the pressure in other parts of the body.

Both the systemic and pulmonary vessels are capable of enormous distention. Experiments have been made which show the surprising fact that large areas may be cut out of the systemic and pulmonary circulations without apparently producing any serious consequences in the circulation. Three-fourths of the pulmonary circulation has been cut off in dogs[1] without reducing the amount of blood delivered to the left ventricle; while the left lung has been tied off in the rabbit without causing a fall in the systemic pressure (Tigerstedt). Tigerstedt suggests that one-half of the pulmonary area may be cut off without any appreciable fall in the systemic blood pressure and suggests as the probable explanation that the vessels of the lung are probably never uniformly filled with blood, there being large areas of the lung relatively empty which are only properly filled when large portions of the pulmonary area are cut out of the circulation. The resistance of the lung is normally small and the blood pressure low.

Effect of Pathological Reduction in Pulmonary Areas.—While experiments such as those mentioned above show the wonderful powers of adjustment in the pulmonary circulation, they must not be interpreted as meaning that a large portion of the lung can be cut out of the circulation with impunity. If the same amount of blood is forced through one-half of the pulmonary area in a given time as should be normally forced through the total area, it can only be done by an enlargement of the vessels and an increase in the power of the right ventricle. That such a condition would be followed by a diminished oxygenation of the blood and in that way eventually prove harmful to the entire organism and result in lessened tissue tone, is to be inferred. This is the condition that we find in advanced tuberculosis. As the disease progresses one area after another is involved in fibrosis and necrosis, and the necessary accompanying condition,—compensatory emphysema—until finally a large portion of one or both lungs is destroyed and the circulation is greatly embarrassed. Under such conditions the systemic blood pressure is not maintained. It becomes progressively lower, regardless of hypertrophy of the right heart and rapidity of the pulse.

Blood Pressure in Tuberculosis.—Blood pressure is lowered early in the disease, although wide departures from the normal are rarely seen except in advanced cases. This shows that the lowering of the systemic pressure is not primarily due to an inability of the right heart to propel the proper amount of blood through the lungs into the left heart, thence into the general circulation, on account of lack of muscular force; although this condition is often finally attained.

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[1] Lichtheim: Die Störungen des Lungen Kreislaufes, Berlin, 1876.

Pike[2] says: "It is my belief that there are at least four mechanisms involved in the maintenance of blood pressure; namely, (1) the vasomotor nerves, whose common point or origin lies in the medulla oblongata; (2) the heart and its nerves—intrinsic, perhaps, as well as extrinsic; (3) the skeletal muscles; and (4) some property of the tissues of the vessel walls, possibly independent of the nervous system, in addition to those properties directly under control."

From the above quotation it is evident that at least three, probably all of the mechanisms which maintain blood pressure are disturbed in pulmonary tuberculosis.

It is generally stated that toxins reduce blood pressure. This is untrue of toxins *per se* unless the toxemia be very severe. Toxins act upon the vasomotor center and through the sympathetic nervous system and produce vasoconstriction. This is one of the causes of rise in temperature which I have discussed in Chapter XXX, Vol. II. The state of toxemia, however, after it has existed for a prolonged time, produces other conditions which have a tendency to lower blood pressure. In early tuberculosis, lowering of the blood pressure is most probably due more to the lessening of the inspiratory act than any other single factor. In advanced tuberculosis many other factors enter, such as degeneration of the heart and wasting of the skeletal muscles.

The effect of tuberculosis upon the heart is many-sided. In early tuberculosis we have not only the effect of the toxins acting centrally through the sympathetics, accelerating its action, but also a peripheral stimulation of both sympathetic and vagus fibers by the inflammation in the lung. The effect of stimulating the vagus fibers is to reflexly slow the heart's action; consequently, through the entire course of pulmonary tuberculosis there is a disturbed innervation, varying greatly according to the extent of the lesion and the degree of activity. Later, changes take place in the heart muscle itself; first, that of hypertrophy of the right ventricle in order to overcome the extra work thrown upon it by the disease blocking off the vessels in the lung; but, later, degeneration of the heart muscle, resulting from toxemia and general malnutrition.

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[2] Nature of Surgical Shock, American Journal of Surgery, October, 1914.

The skeletal muscles, while not particularly disturbed at first, sooner or later lose their tone, partaking of the general wasting of the tissues throughout the body. The loss of support to the vessels arising from decreased tissue tone must, of necessity, greatly interfere with their ability to maintain their normal tone, and it would not be at all beyond reason to suspect that vessel walls are weakened, the same as all other tissues of the body.

There is no doubt that blood pressure, lower than normal, must exert an unfavorable influence upon the patient. In order to maintain proper conditions for metabolism to go on, a certain normal pressure in the circulation is demanded. In tuberculosis, to the extent that this normal is reduced, there must be interference with nutrition. The effect of the various factors operating to alter pressure is to cause a wide departure from the normal in the blood content of various groups of vessels. The blood remains stored up in the venous reservoirs of the body, particularly in the liver and splanchnics, while the arteries are relatively empty.

The effect of low pressure may be inferred from the following quotation from Pike:[3] "Whatever the immediate cause of the low blood pressures may be, it may well become part of a vicious circle. Even though the blood be well oxygenated by artificial respiration, and the heart be beating regularly, a previously damaged portion of the central nervous system: e. g., the brain, after subjection to anemia, does not recover as long as the systemic blood pressure remains low. Nor does this fact need surprise us when we remember that, among the other relatively constant conditions of the mammalian body, a blood pressure varying but a few millimeters under the various conditions of activity from day to day or year to year is an important item. Any considerable variation from this usual level is strong presumptive evidence of abnormal processes involving other mechanisms than that of circulation."

The failure of the heart to meet the requirements of exercise, particularly sudden strain in advanced tuberculosis, can easily be explained. The extra amount of blood required for the mus-

[3] Nature of Surgical Shock, American Journal of Surgery, October, 1914.

cles during exercise is derived from the splanchnic area and owing to the lessened inspiratory act, the conditions are such that it cannot be readily delivered to the right heart.

Blood pressure is low in all stages of tuberculosis, although the readings of different observers show no uniformity on account of individual variability. The degree of fall in pressure is modified by many factors, but is progressive as the disease advances. My findings in blood pressure are well represented by the statistics of 162 patients examined and compared with 20 non-tuberculous individuals.[4] The readings were made by the Stanton sphygmomanometer.

	Sys.	Dias.
20 non-tuberculous individuals	120	108 mm. Hg.
11 patients in I Stage	106	78
21 patients in II Stage	108	81
130 patients in III Stage	103	75

While in this table the pressure in Stage II was higher than that of Stage I, this is purely accidental. Had the number of patients been larger the pressure in Stage I would have undoubtedly been somewhere between the reading of 106 and that of 120 noted for the non-tuberculous. The only fact which I wish to illustrate is that the pressure is lower than normal and that it progresses with the disease. It is not uncommon in far advanced cases to find instances where the pressure falls to 90 mm. Hg. and at times below this.

Extremely low pressure is associated with a very low tissue tone. Patients having very low pressure feel relaxed. They lack endurance, are easily fatigued, sometimes on the slightest exertion, even though not out of bed. On motion they often feel dizzy and faint. Poor appetite and digestion are often accompaniments. We meet this when there has been an extensive destructive process in the lung, associated with marked interference with the inspiratory action of the thorax, and consequently a marked splanchnic congestion, a high grade of toxemia and a general wasting. The low pressures of advanced tuberculosis are approaching the point of syncope and are inconsistent with feelings of well-being and incompatible with physical exertion.

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[4] The Effect of Tuberculosis on the Heart, Archives of Internal Medicine, October, 1909.

These low pressures are benefited by such lines of treatment as abdominal massage; treatment which will produce a deposit of fat in the abdomen; adhesive straps or belts which increase the intra-abdominal pressure and aid in forcing the blood from the splanchnic areas toward the heart; also by such remedies as have a direct tendency to raise blood pressure such as digitalis, citrate of caffeine and suprarenal extract. In the use of digitalis, larger doses should be employed than usually given. I not infrequently give fifteen minims or more of a standardized tincture three times a day for two days, then drop to ten minims for a period of four or five days; I then omit the remedy for two or three days and resume the same as before, keeping up its use over a considerable time.

It has been noticed that a rise in blood pressure and clinical improvement go hand and hand. Some observers have attributed the improvement in the pulmonary condition to the increase of blood pressure. This is a factor in the increased well-being of the advanced case as just mentioned, but it cannot be considered as standing in a causative relationship to the cure and arrestment of the tuberculous process. In early tuberculosis when normal. The rise in pressure stands as a result, not as a cause of the pulmonary improvement. When increased pressure appears with improvement it is due to increased nerve stability, improved inspiratory action, improved nutrition, and better heart action. It cannot be denied, however, that the increased pressure reacts and further increases the patient's feelings of well-being and in the infiltrations in the lung disappear, nerve tone improves and the normal inspiratory excursion is restored wholly or almost wholly, the blood pressure rises and attains or approaches the that way furthers his improvement.

Small Heart and Arteries.—In Chapter XI, page 301, I discussed the question of the small heart and suggested elsewhere[5] that it is probably the result of an embarrassed inspiratory act through which the heart is required to accustom itself to a decreased blood content, likewise a decreased output. Benecke, as well as other pathologists, has shown that the same conditions obtain in the arteries, the arteries being narrower than normal. It can be seen how this cause would operate to produce narrowing of the arteries, as well as a reduction in the size of the heart. That the malnutrition of the late stages leads to an atrophy of the heart muscle is well established; but this is entirely apart from the small heart which has been spoken of by clinicians and which is found in early tuberculosis. Bohland[6] in discussing this question, states that Reuter[7] and Hirsch[8] and Kersten[9] proved the presence of the small heart by orthodiagraphic examination. He says: "Both found in the majority of tuberculous subjects (62.5-88 per cent) hearts which were too small; in fact, Kersten found hearts below normal in size in 62.5 per cent of well nourished patients." Not only were Kersten's patients well nourished; but a large number of them were in the first stage of tuberculosis.

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[5] The Small Heart in Tuberculosis: A Suggested Physiologic Explanation, Journal American Medical Association, April 17, 1915.

Hypertrophy of Right Ventricle.—Tuberculosis presents several conditions tending to disturb circulation. On the part of the pulmonary circulation we have the obstructions in the vessels supplying areas of lung tissue of greater or lesser extent, either by the infiltration and collateral inflammation, or the destruction of lung tissue. We also have the embarrassment due to the encroachment upon the vessels which results from compensatory emphysema.

It is evident from these conditions that, at least for a long time the circulatory burden in tuberculosis is upon the right heart. There is an obstruction of a permanent character to the circulation in the pulmonary vessels. This calls for permanent extra work upon the right heart. In order to measure up to the demand the muscle hypertrophies. The right heart bears the brunt of the disease and ultimately is the deciding factor between life and death.

Thickening of Arteries in Tuberculosis.—Examination of the arteries in chronic tuberculosis shows them to be thickened.[10]

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[6] Handbuch der Tuberkulose, Brauer, Schröder und Blumenfeld, Johann Ambrosius Barth, Leipzig, 1915.

[7] Dissert. München, 1884.

[8] Deutsches Archiv fur klinische medicin, Bd. c.

[9] Deutsche medizinische Wochenschift, 1911.

[10] Pottenger: The Effect of Tuberculosis Upon the Heart, Archives of Internal Medicine, October, 1909.

The analysis of 162 patients examined with reference to the condition of the arteries showed the following result:

	DURATION OF ILLNESS FROM FIRST DEFINITE CLINICAL SIGNS		
CONDITION OF RADIALS	LESS THAN 1 YEAR	1 TO 2 YEARS	MORE THAN 2 YEARS
Palpable	14	20	60
Non-palpable	14	21	33

These results were based on the examination of the radial artery, and were obtained after stripping the radial from below upward with the second finger of one hand and above downward with the second finger of the other hand, and then feeling the radials, emptied of blood, with the forefinger. Unless the precaution of stripping the vessel of blood is taken, the result will be unsatisfactory.

Later examinations confirm these earlier observations.

The cause of the thickened arteries is most probably a reflex trophic change due to toxemia. Support for this theory is seen in the effect of other toxemias in producing the same condition, and the fact that this condition is more common in chronic cases where the causative factors have had a longer time to act.

Tuberculous Lesions of the Blood Vessels.—Tuberculosis may affect the blood vessels either from within or from without, and the lesion may be of all degrees of severity from that of very slight trauma to necrosis. Sometimes the infection is followed by an opening in the vessel wall and hemorrhage; sometimes an infiltration results which goes on to repair; and sometimes an endarteritis with obliteration of the lumen occurs. It seems probable that many of the hemorrhages which we have in tuberculosis, particularly those where only small amounts of blood are expectorated, may be sanguineous exudates due to the injury of vessels resulting from the action of toxins.[11]

Bacilli invade the endothelial cells, give off toxins which attract leucocytes and thus form tubercles in the walls of the vessels. Miliary tuberculosis is a tuberculosis disseminated by way of the blood vessels in which many bacilli have escaped into the blood stream and found lodgment in small vessels. It is from just such lesions as those here described that the bacilli which produce acute miliary tuberculosis come. The capillaries are most commonly affected, while small veins and arteries are less commonly so. Tubercles may be found in the walls of any of the vessels of the body, including the aorta. This being true, it is surprising that miliary tuberculosis is not a more common infection than it is. That it is not, is probably due to the fact that the vessel is often occluded by the inflammatory process so that bacilli do not get into the blood stream, and second that protective cell sensitization and antibodies reduce the virulence of or destroy the bacilli which gain entrance to the blood stream before they find lodgment in the tissues, in those patients in whom infection has been present long enough to build up a specific immunity.

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[11] Pottenger: Some Observations on the Classification and Treatment of Hemoptysis, American Journal of Medical Sciences, June, 1914.

Difficulties in Examining Hearts in Tuberculosis.—The examination of the heart in advanced pulmonary tuberculosis offers many difficulties. The landmarks which have been learned in normal chests no longer hold because the heart is no longer surrounded by normal tissues; and, itself, is often displaced.

One does not always find, I might say in chronic advanced tuberculosis he rarely finds, the point of greatest intensity for the pulmonary valve at the second interspace to the left of the sternum, or the aortic valve at the second interspace to the right of the sternum, or the mitral at the apex which is usually found in the fifth interspace somewhat internal to the mammary line. In the shiftings of position which the heart undergoes as one portion of the pulmonary tissue wastes and contracts and other portions enlarge, the site for the auscultation of these valves shifts also. In order to be able to examine the valves it is essential to first outline the heart as a whole. The outlining of the area of absolute heart dullness is of comparatively little value in advanced tuberculosis. The information desired can be obtained only by knowing the accurate outlines of the organ as it lies in the chest. It must be remembered that it is the most movable portion of the mediastinum and as such may be shifted to the right or the left as is demanded by the intrathoracic compensation.

The points of greatest intensity for auscultating the pulmonary and aortic valves may both be on the left of the sternum in marked left displacement or on the right of the sternum in marked right displacement, or in any position between these two, as shown in Figs. 41 and 42, Chapter XI.

The visible pulsation on the chest wall must not be taken for the apex. Quite often it is produced by the hypertrophied right ventricle as mentioned above and careful observation will show that the intercostal space draws inward as the ventricle contracts, instead of being thrust outward as it would be if the impulse were caused by the apex. Under such circumstances the apex will be found a considerable distance to the left of the point of pulsation.

The inferior border of the heart is also at times lower than normal. Especially is this true in long-chested, enteroptotic individuals, also in those who have forced the diaphragm down by emphysema and at times when tuberculosis develops in individuals who are getting along in years, or who, because of their disease, age prematurely. Suspensio cordis is not uncommon in such cases.

It is important to bear in mind that the valve sounds are greatly influenced by their surroundings, consequently they cannot be compared as they can be in individuals who have no change in the pulmonary tissue. While the valves at the base of the heart are usually covered, when contraction of the pulmonary tissue occurs at one apex and this is followed by enlargement and compensatory emphysema of the other lung, one valve may be directly against the chest wall while the other has an additional thickness of pulmonary tissue between it and the chest wall. Listening to these two valves under such circumstances with reference to comparing their intensity, can give none but erroneous information.

Consolidation of lung tissue, emphysema and cavities affecting the tissues adjacent to the valves also affect the character of the tones.

Organic Heart Lesions and Tuberculosis.—If we accept the idea that clinical tuberculosis is an accident which may happen to any member of the human family, we will not be far from the truth; and, further, we will be ready to look for about the same general complications that we would expect in the same number of non-tuberculous individuals, unless there be some specific reason why such complications militate against tuberculosis.

It is quite generally accepted that all such organic heart lesions as from their nature produce hyperemia of the pulmonary tissue, are antagonistic to the development of pulmonary tuberculosis. This does not, however, preclude the possibility of such infection taking place, or the possibility of such an infection developing into active clinical disease when the infection has occurred; for all such protective conditions must be looked upon as being more or less relative.

I have met with a number of instances of such heart lesions in tuberculosis and my experience with them has been unfavorable; so, while they may render a relative resistance to the bacillus, yet I cannot feel that the hyperemia produced is a factor which can be at all compared with the aid in arrestment which comes from a normal heart.

Heart Bruits.—The heart tones in tuberculosis are often impure. Sometimes a distinct bruit is present which partially or wholly displaces the heart tone. Sometimes it is heard best on ordinary inspiration, sometimes it is eliminated by forced inspiration.

The character of these bruits varies, but for the most part they are soft. They cannot be positively separated from organic heart bruits. In fact, some writers believe that all bruits which take their origin from the valves are produced by some abnormal vibration of the valves or the adjacent vessel walls. Aside from the bruits which cannot be separated from the heart valves there are others which are unmistakably caused by pleuropericardial adhesions and others due to kinking and encroaching upon the lumen of the vessels by the pathological changes present.

Degeneration of Heart Muscle.—The hypertrophied heart is able to meet the demands of the circulation at first; but, as the changes in the lung become more widespread, a time comes when any extra exertion is met with difficulty and cardiac weakness of a greater or lesser degree manifests itself. This is the greater when the disease is not only widespread but active; for, during this time malnutrition is increasing and poisons are being poured out into the tissues, both of which cause degeneration of the heart muscle along with other tissues.

Strangely, the heart in tuberculosis has not received as much consideration as it deserves. This has been due to our narrow view of tuberculosis, looking upon it as a disease of the lungs, when, in reality, it is an infection of the pulmonary tissue from which, at least in the advanced stages, every organ and cell of the body is injured. The heart comes in for an unusual amount of disturbance, both in its texture and function.

I have spoken of the small heart which is found in tuberculosis, even in the early stages and have given a physiological explanation for it. There is also an atrophy of the heart in tuberculosis which occurs as a late manifestation being a part of the general malnutrition and muscle wasting.

The result of prolonged toxemia in tuberculosis finally shows upon the heart muscle. As Satterthwaite[12] says: "We can now say that all toxemias, acute or chronic, cause dyscrasias and hyperemias if long continued or severe, and other conditions to be mentioned later, produce definite morbid changes in the heart walls, evanescent or permanent, as the case may be." This fact calls for special attention to the heart in dealing with this disease. All things else being equal, the patient with the good and well cared for heart has a decided advantage in his struggle for overcoming tuberculosis and particularly advanced tuberculosis.

The subject of valvular lesions is of comparative insignificance to the condition of the myocardium itself in tuberculosis; for the majority of deaths in this disease are due to an exhausted heart muscle. The changes in the muscle substance are not as evident in a slow going toxemia like tuberculosis as they are in a disease in which the toxemia is of a very high grade such as diphtheria; yet, they are no less a factor in determining the outcome of the disease. The changes in the muscle substance in tuberculosis are general, as a rule; and the ultimate effect, no less than in acute toxemia, is to interfere with the contractility of the muscle, reducing the heart power.

Unfortunately, in advanced tuberculosis, there is no way of

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[12] Cardio-Vascular Diseases, New York, 1912.

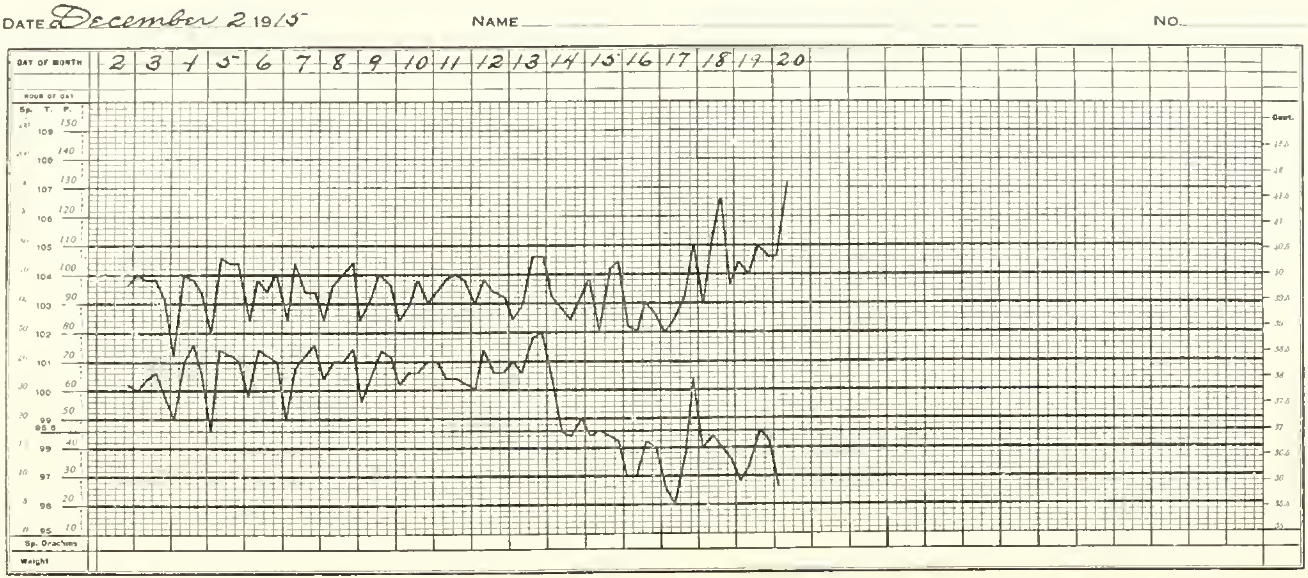

Fig. 38.—Illustrating terminal dilatation of the heart in a patient who was suffering from advanced fibrocaseous tuberculosis. It will be noticed that on the 14th the curves of temperature and pulse began separating, the temperature dropping and the pulse increasing in frequency.

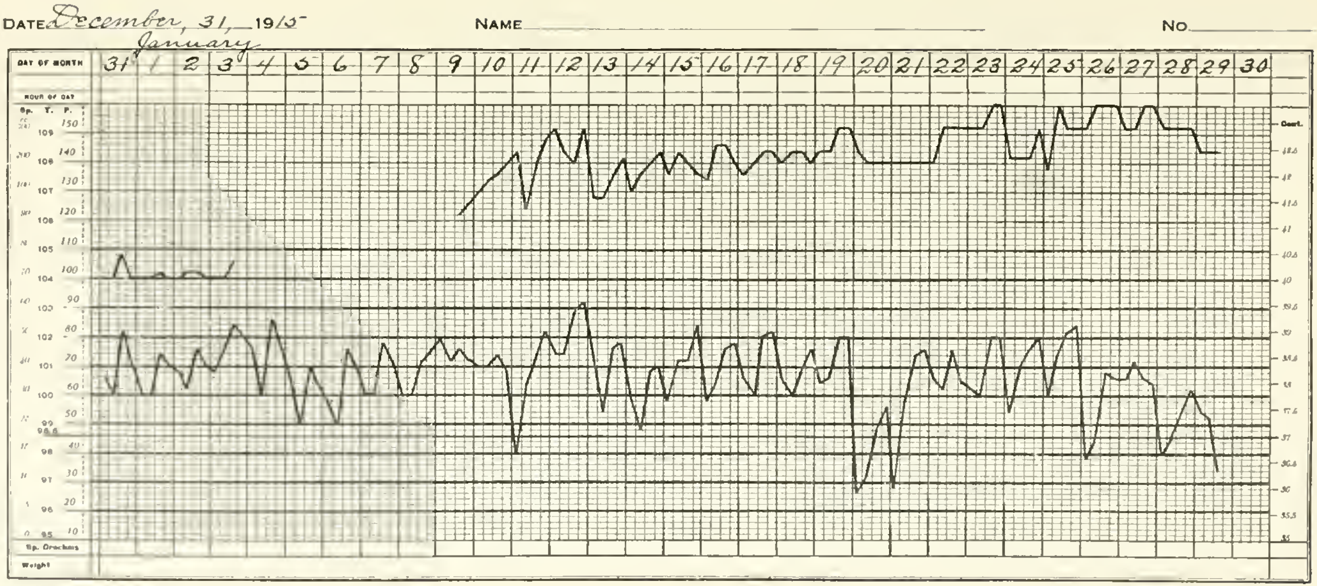

Fig. 39.—Illustrating terminal dilatation of the heart in a patient suffering from chronic caseous tuberculosis. Dilatation began to manifest itself during the early days of January. The pulse in the case went unusually high, reaching 160. The fall in temperature was not as marked as is usual; although it is quite evident during the last four days of life.

relieving the work which is thrown upon the right ventricle. Whatever embarrassment exists as a result of blocking off and obstructing the flow in the blood vessels of the lung is permanent; so, the only way of affording relief is through increasing the strength of the heart itself, and guarding it against all unnecessary strain.

Clinical Evidence of Failing Heart.—The symptoms which first appear when the heart begins to weaken under the strain in tuberculosis are rather vague and easily go unrecognized. It is much easier to look back and ascribe to them their proper significance than it is to recognize them at the time. The symptoms which I have noted most commonly are, nervousness, anxiety, insomnia, lagging appetite, and at times, poor digestion. These symptoms can be caused by so many conditions that unless one has the heart in mind the true cause will most likely be overlooked. Breathlessness usually comes later.

The pulse at the same time becomes soft and weak and the heart tones become weak on auscultation. But this latter characteristic is not always readily discovered, because the sounds may have been weak previously. These symptoms are often present before any marked dilatation can be determined; this, with precordial pain usually being a later manifestation. Some increase of the pulse rate usually accompanies the early signs of weakened heart, but not the progressively rapid pulse noted at the time of acute dilatation. On examination an enlargement of the heart from left to right is made out on palpation and percussion when dilatation has occurred.

Figs. 38 and 39 show the way in which the pulse and temperature curve separate in terminal dilatation of the heart. As the heart becomes more and more unable to do its work it contracts less forcibly and more rapidly, the body becomes less and less able to react and so the temperature falls lower and lower until death results. See pages 246 and 247.

Myocardial weakness, as it affects the right heart, produces a very different picture from weakening of the left heart. Dyspnea is not a very prominent symptom when the right heart is at fault, except when the heart is called upon to measure up to an extra exertion. It is a marked symptom of weakness of the left heart, however, even when the patient is at rest. Stasis, in the systemic veins, however, is regularly present in weakness of the right heart, but not regularly present in weakness of the left. Pulse disturbances are present in both. When dyspnea and venous stasis are both present, together with pulse disturbances, it is evidence of weakness of the entire heart. Death from tuberculosis, being usually a right heart death is peaceful and painless.

Treatment of Failing Heart.—Not being able to relieve the load on the right ventricle we can only strengthen it and make as little demand upon it as possible. In order to accomplish the latter, rest in bed should be enforced. It will be recalled that the amount of oxygen made necessary by arising from the recumbent to the erect position is doubled and the amount to start going is quadrupled. This can only be furnished by the heart increasing its output of blood in a given time. Consequently, rest in the recumbent position is one of the most valuable aids to such a heart, for it relieves it of all removable load.

For the heart muscle itself I have great confidence in the use of digitalis and citrate of caffeine. If the early symptoms mentioned above are recognized and the patient is put in the recumbent position and given digitalis in proper doses, the crisis can often be averted. Digitalis strengthens the heart's contractions and in this way probably affords better nutrition to the organ; but, whether this is the explanation or not, I have great confidence in its use. If digitalis is to be used, it should be used in doses sufficiently large to produce an effect. I use a standardized tincture in doses of x to xx minims three times a day until the pulse improves or the physiological effect is reached. This is generally shown by a nausea or irregularity of the heart. I then discontinue the remedy for three days and then repeat again, giving the remedy three or four days and then withholding it for a like period. Citrate of caffeine can be given in iii grain doses every four hours and continued as long as necessary.

The patient should be fed nourishing food in concentrated form. The bowels should be kept free to relieve any toxins that can be removed through the alimentary canal. The important thing in the handling of these cases is to recognize them early, and to treat them properly. These degenerative changes in the heart muscle may threaten the patient's life and yet the heart may recover itself as the patient improves.

CHAPTER X.

THE DIGESTIVE SYSTEM IN TUBERCULOSIS.

General Observations on Nutrition.—How different our conception of nutrition and its problems since we have come to learn that digestion is more than the dissolving of food by salivary, gastric, and intestinal ferments. Substances of all kinds enter the stomach as food, substances which, if absorbed in the state in which they are ingested would completely disorganize the entire cellular system. The function of the gastrointestinal juices is to break down the many compound substances which are eaten as food and to reduce them to simple substances which the body can utilize. In the form in which they are ingested many of them are not foods, although they contain substances which may be converted into foods and utilized in building up cells and furnishing energy for running the human machine.

The protective action of the gastrointestinal ferments is a very important part of its digestive function. Our admiration for nature's protective program for man is greatly increased by our better understanding the protective plan of the digestive tube.

The heterogeneous masses which are ingested are broken down and split up into substances which the organism can utilize and those which it cannot. The latter, if absorbed, would be harmful. The former, in order to be properly utilized must be carried to certain structures to be further elaborated. The cells of the mucous membranes of the digestive tube are endowed with such defensive powers, we almost feel like saying selective intelligence, that they pick up those particles that the body cells can utilize and refuse to take those which it cannot use. Not only this, but different cells pick up different substances; thus the fats are absorbed and carried through the lymphatics to the blood, while the substances derived from carbohydrates are taken up by the blood and carried to the liver. Should any substance which is not in harmony with the tissues gain entrance to them, the cells with which they come in contact outside of the digestive tract, form defensive ferments and split them up into substances which may be utilized as food and other substances which cannot. These latter are then acted upon further and further until they are rendered harmless, if that be possible; or, if not, until they are excreted from the body, or gain mastery over it. Abderhalden's discussion of the defensive ferments produced by the cells clarifies some of the problems of human existence and sheds light upon questions of maintaining the body cells in a state of equilibrium.[1]

Nutrition in Tuberculosis.—The question of nutrition in the tuberculous, the same as in the healthy, comes far from being wholly a question of what or how much a patient eats, but has to do with the entire question of metabolism. Faulty nutrition may be due among other things to pathological conditions in the gastrointestinal tract, such as disturbances in secretion, or motility, or any acute inflammatory disease affecting the canal; errors in diet consisting of either eating wrong food or food in improper quantities, or in an improper manner; degenerative changes in the body cells which interfere with either the absorption of food particles from the alimentary tract or the proper exchanges between the blood and the tissues; pathological conditions in the blood itself; interference with the rapidity of blood flow; and a general lack of harmony in the function of the various important systems of the body.

Pathological changes in the gastrointestinal tract, as they are found in tuberculosis present several different conditions, the ultimate effect of which in each case is the interference with proper absorption of nutriment. An almost constant condition in the alimentary tract in tuberculosis is a passive congestion. Resulting from the insufficient motion of the diaphragm and other inspiratory muscles there is always more or less splanchnic congestion present, the degree varying with the amount of disturbance in inspiration. This lessening of the inspiratory act causes the blood flow to be retarded through all the abdominal organs and favors a general blood and lymph stasis, which interferes with the secretory function of the digestive glands, the motor function of the gastrointestinal walls, and the absorption of nutriment from the alimentary canal. The amount of disturbance due to these conditions varies from that which is scarcely discernible clinically, to a very markedly altered function in some patients who are suffering from more advanced lesions. Not only do we have a passive congestion as a result of deficiency in the respiratory act, but also as a result of nervous influences and weakened heart action, the latter particularly in advanced tuberculosis.

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[1] Abderhalden: Defensive Ferments, William Wood & Co., New York, 1914.

Displacement of the various organs is also very common in tuberculosis. This is particularly true in the advanced disease as a result of malnutrition and the consequent absorption of the intra-abdominal fat, the general weakening of the musculature of the abdominal wall, and the wasting of the tissue supports of the various intra-abdominal organs. Aside from this we must also consider the effect of the pathological process above the diaphragm. The diaphragm is often displaced and pushed downward by compensatory emphysema and at times by pleural effusions and pneumothorax. Constant coughing also has an influence in forcing the abdominal organs downward. Probably, what is of greater importance than the ptosis itself in these cases is the reduced intra-abdominal pressure under which the abdominal organs are compelled to work as a result of this loss of tissue and tissue tone.

This ptosis affects, at times, all the abdominal organs. In advanced conditions it is not uncommon to find the stomach resting on the pubes; the cecum and transverse colon, together with the small intestine wedged tightly into the pelvic cavity, and the lower pole of the right kidney below the level of the umbilicus. A dilated cecum is also frequently found. These ptosed organs, however, often functionate well and show no symptoms directly referable to the ptosis itself.

Atony and dilatation are very common in advanced cases.

The gastrointestinal tract is subject to many reflex disturbances. In some the afferent impulse travels from the lung over the vagus. Others come from the surface of the body through the spinal nerves.

The efferent impulse is transmitted through the gastrointestinal branches of the vagus and sympathetics, respectively to the vessels, glands and muscles of the organ. I have recognized this gastric vagus reflex from the lung in several instances of hyperacidity which were increased by the focal reaction following large doses of tuberculin. I have also seen it in numerous instances during cavity formation in the lung, where, as a result of irritation of the pulmonary branches of the vagus, hyperacidity, reflex vomiting, and spasticity of the colon were produced.

Neither must we forget the effect of toxins on the gastrointestinal tract. These have a two-fold action. They act centrally through the splanchnics, exercising an inhibitory influence upon digestive activity in general; and, particularly late in the disease, cause degeneration of the cells of the organ.

Tuberculosis of the various portions of the intestinal tract and of the liver, pancreas, and spleen, must also be considered as part of the pathology which must be reckoned with.

The Digestive Tract and the Vagus and Sympathetic Nervous Systems in Tuberculosis.—The part which the nervous system plays in the production of symptoms on the part of the gastrointestinal canal during the course of clinical pulmonary tuberculosis, is a very important one. As most internal viscera are innervated by both the parasympathetic and sympathetic divisions of the vegetative system, and as these divisions antagonize each other in every viscus in which they meet, as described more fully in Chapter VII, it behooves us to study carefully the effect of central or peripheral stimulation of either of these systems which might occur as a result of the inflammation in the lung.

As I have shown elsewhere[2] and discussed in Chapter VIII, the syndrome of toxemia as expressed peripherally is that of general sympathetic action. Its effect is that of a central stimulation of the nervous system plus a stimulation of the sympathetic structures peripherally.

No matter what the source of the toxemia, the expression is the same.

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[2] The Syndrome of Toxemia: An Expression of General Nervous Discharge Through the Sympathetic System, Journal American Medical Association, Jan. 8, 1916, vol. lxvi, pp. 84 and 85.

On the part of the gastrointestinal canal, toxemia manifests itself in inhibition of action. This is the system of nerves which diminishes the secretion of the salivary and mucous glands, relaxes the stomach, inhibits gastric, intestinal, hepatic, and pancreatic secretion, relaxes the intestinal wall and decreases the motility of the gut. When toxemia is present, the sympathetics are centrally stimulated by the toxins and peripherally by the extra adrenin which results from the splanchnic stimulation, and we have a chain of symptoms, varying, of course, according to its severity, such as; a decrease in appetite, coated tongue, diminished gastric and intestinal secretions, and a deficient gastric and intestinal motility resulting in stasis and constipation.

In tuberculosis the degree of toxemia varies greatly with the character of the disease; so do the symptoms on the part of the gastrointestinal tract. Sometimes they are very severe and again they are negligible. Sometimes, even when a marked degree of toxemia is present, no noticeable symptoms of gastric or intestinal origin will be noted. This can be explained on a rational basis by bearing in mind that tuberculosis is an inflammation in the lung, which irritates the pulmonary nerve endings belonging to the vagus; and, when producing toxins, stimulates centrally the sympathetic division of the vegetative system; and these being antagonistic in their action in the gastrointestinal tract, no disturbance in equilibrium results.

Peripheral sympathetic stimulation, like central stimulation of the same division of nerves may possibly cause inhibition of function in those organs which come in the path of its reflex action. Whenever a toxemia stimulates the central nerve cells of the sympathetic in pulmonary tuberculosis, the inflammation at the same time stimulates the peripheral filaments of the vagus. The parasympathetics being the division of the vegetative system which promotes intestinal action, when stimulated, oppose the inhibiting influence of the sympathetics and in many instances wholly counteract them.

Appetite.—The appetite is decidedly variable in tuberculosis. During the periods of general depression, such as those which are manifested by discontent, discouragement, anxiety, fear and during pain; and also during periods of toxemia, the appetite is usually diminished. At other times, it may be normal, or even above normal. The appetite is very important in tuberculosis, both because of its influence on the amount of food taken and upon the digestion of the food after ingestion.

During the entire period when toxemia is present and during the periods marked by the depressive emotional states that accompany or occur during the disease, the appetite is usually very seriously lessened. At times, it is impossible for the patient to eat sufficient food to nourish him. Owing to the lack of appetite, mastication of food, which is so essential to the proper division of the particles and also to its insalivation, is imperfectly carried on. Often it is necessary that the great bulk of the food eaten be of such a nature that it can be swallowed rather than chewed. The result of this is that an extra burden is thrown upon the gastric and intestinal functions. While normal gastric juice is able to take care of protein food, whether it be coarsely or finely divided; yet a lack of proper insalivation, particularly of the starchy food, has a very important inhibitory action upon gastric digestion.

Maxwell[3] shows experimentally that substances in suspension or in colloidal solution have the power of absorbing enzymes, thereby inhibiting their activity. He has also shown experimentally that unboiled starch administered in intact grains does not hinder the action of pepsin; but when starch has been boiled and left in colloidal form, it seriously interferes with digestion by the absorption of the proteolytic enzyme. Thus, the time interval for the peptic digestion may be increased fourfold in the presence of a 2 per cent starch solution.

From this the conclusion is drawn that all cooked farinaceous foods, such as rice, sago, potato, bread, and oatmeal, interfere with peptic digestion unless they are thoroughly insalivated. This work emphasizes the importance of chewing farinaceous food thoroughly. Salivary, gastric, and intestinal digestion are more or less closely related; yet each has its own particular function and, unless carried on properly, interferes with, or throws an extra burden upon the process next lower.

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[3] The Relation of Salivary to Gastric Secretion, Biochemical Journal, 1915, vol. ix, p. 323; quoted editorially in the Journal of the American Medical Association, vol. lxvi, March 4, 1916.

Disturbance on the Part of the Stomach.—Our idea of the stomach and its importance as an organ of digestion is very different from what it was a few years ago. We cannot lose sight of the fact that it may show many disturbances in function, due to action outside of itself. While the stomach may be looked upon as a reservoir for food, retaining it until it is ready to be passed on into the intestine; yet certain important changes take place in the food during its stay in this reservoir which are preparatory to further digestion; and it is necessary that this organ have proper secretory and motor power if normal relationships are to be maintained in the remaining portion of the digestive canal.

The importance of the quality of the gastric contents as discharged into the duodenum cannot be overestimated if our newer theories of secretory and motor control of the gastrointestinal tract are to be held.

The hormone theory as it affects digestion presupposes a nerve stimulus awakened by the smell, taste, and intake of food. Through this the gastric hormone is set free into the blood and finds its way to the secretory glands of the gastric mucosa and stimulates them to the production of gastric juice. When the acid gastric contents are discharged into the duodenum, they stimulate the formation of another hormone, which passes into the blood and in its circulation exercises its selective action upon the secretory glands of the intestinal wall, pancreas and liver, setting free the digestive ferments of these various organs. Henry Harrower[4] has discussed this matter in a monograph well worthy of perusal. The hormone theory, like all other contributions which have added to our knowledge of the function of the gastrointestinal tract, and which have pointed the way to a better understanding of the nervous derangements which are found in our every day practice, emphasizes the fact that disturbances of digestion cannot be considered as belonging to any one portion of the tract alone, but to it as a whole.

We must still recognize the alterations which occur in the gastric secretion because they are very important in their bearing upon further digestion. If we have abnormal secretion in the stomach in tuberculosis, we are apt to have abnormal secretion in other parts of the gastrointestinal tract from the same cause; abnormal motility, also; because the motor function of the intestinal canal depends on the stimulus received from the acid contents of the stomach. In early tuberculosis increased acidity, probably of reflex origin, is quite common. A general, increased tone is present affecting all of the vagus branches as suggested by Eppinger and Hess.[5] During the late stage of the disease, especially when toxemia is marked, a deficiency in hydrochloric acid is the rule, although this condition often alternates with normal amounts of acid, or with a hyperacidity. The relaxed conditions produced by toxemia and its action upon the sympathetics and by the general degeneration present, contrary to what we might think, are not always accompanied by a deficiency of hydrochloric acid. Not infrequently do we find a hypersecretion during these periods. This can be explained on the ground that the reflex stimulation of the vagus is stronger than the combined reflex and central stimulation of the sympathetics. Inasmuch as control of the pylorus depends to a considerable extent upon the acidity of the secretion, as it passes from the stomach into the duodenum, we must recognize the importance of the quality of the gastric juice because of its effect on the entire canal.

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[4] Practical Hormone Therapy, Baillière, Tindall and Cox, London, 1914.

At times we see, especially in the later stages, a decided increase in the amount of secretion which remains in the fasting organ. This may have either a normal, a deficient, or an increased percentage of acid. This condition is often accompanied by more or less troublesome symptoms. It seems to be associated often with atony and dilatation.

Atony and dilatation of the stomach are usually associated with a downward displacement of the organ. This dilatation may be due to errors in diet, overfeeding, altered secretion accompanied by abnormal gas formation, obstruction of the pylorus, or to a general degeneration of the cells of the wall of the organ (I have seen cases postmortem where the stomach was dilated to such an extent that its capacity was three or four times its normal). Hypochlorhydria is often found in tuberculosis in patients in whom there is a generally decreased vagus tonus. In such, and usually in others, there is a deficiency of acid during times of acute toxemia owing to the fact that the toxins stimulate the sympathetics centrally and inhibit the gastric secretion.

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[5] Die Vagotonie, Sammlung, Klinischer Abhandlungen, von Noorden, Heft. 9 u. 10. Berlin, 1910.

Hypochlorhydria.—There is a certain class of individuals who naturally have slow digestion. It would be manifestly erroneous to expect the spare asthenic to digest as rapidly and as well as the sthenic or hypersthenic individual. When those of the hyposthenic and asthenic type develop tuberculosis the question of nutrition usually becomes a more serious problem than it does in the stronger individuals. The toxemia of tuberculosis, added to the already slow digestion makes a serious complication.

Since toxemia and states of anxiety, disappointment, and discouragement, as well as painful conditions when present, cause a hyperirritability in the entire vegetative system, they disturb the secretory and motor functions of the entire gastrointestinal canal; and, as can be seen, these have a tendency to retard digestion and produce disturbed motility, with stasis of the bowel contents. This is again followed by absorption of poisons from the alimentary canal which further depresses the digestive functions. The effect of these factors is greater upon those with naturally slow digestive function than upon those with function normal or above normal.

When suffering from hypochlorhydria, if only temporary, it is well to urge patients not to eat large quantities of food until this condition is corrected. The best foods under these circumstances are those which have rather a stimulating effect on the gastrointestinal tract. Meat extracts, strong consomme, meat itself and dry toast, well chewed, stimulate secretion. Dilute hydrochloric acid, 5 to 10 drops taken after meals is of considerable value. The following prescriptions I have used to advantage:

℞	Acid hydrochlor dilut	25.00
	Glycerin	25.00
	Pepsin scales	50.00
	Aq. dest. q. s. ad.	100

M.

Sig.: 1 to 2 teaspoonfuls in water one-half hour after meals.

R	Tinct. nux vom.	12.00
	Tinct. cinchon. q. s. ad.	100.00

M.
Sig.: One teaspoonful in water 20 minutes before meals.

On the other hand, if the deficiency is chronic, then the patient must be fed liberally, using a diet which will stimulate secretion and favor regularity of bowel movement. At the same time, hydrochloric acid should be administered. General measures for improving the patient's metabolism are of the greatest importance.

Hyperchlorhydria.—Hyperchlorhydria, contrary to the general belief, is quite common in pulmonary tuberculosis. As Eppinger and Hess have well pointed out, there is a large class of individuals which may be said to have naturally parasympathetic hyperirritability. The vagus nerve is the nerve which presides over secretion and motility for the gastrointestinal canal; consequently the vagotonic would naturally have an ample acid secretion. Those who are not vagotonic naturally could be rendered so by stimulation of the parasympathetics alone or both parasympathetic and sympathetic systems, but in such a manner that the parasympathetic stimulation predominates over the sympathetic.

During toxemia in pulmonary tuberculosis, as previously mentioned, the sympathetic system is stimulated centrally by the toxins and peripherally by the inflammation in the lung and adrenin which is increased by the central sympathetic stimulation; and, the vagus is stimulated peripherally. As a result of this, sympathetic stimulation is nearly always predominant during toxemia, at least to a certain extent. As soon as the acuteness of the toxemia passes away, and the central stimulation of the sympathetics lessens or ceases, then in the great majority of cases of tuberculosis, the patient passes into a condition of increased parasympathetic tonus. His appetite improves and his digestive powers are even above normal. A state of hyperchlorhydria often exists under these conditions. As a rule it is not sufficient to cause discomfort; at other times it is necessary to afford relief to the patient. Sometimes hypersecretion only is present without an increase in the amount of acid.

Increased acidity is often accompanied by a stasis of the intestinal contents and a spastic constipation, both being due to increased parasympathetic tone. The explanation of this condition is that the peripheral stimulation of the vagus by the inflammation in the lung results as a rule in the vagus impulse being stronger than the sympathetic stimulation and the conservative forces predominating over the inhibiting influence of the sympathetics.

In case of a marked hyperchlorhydria all stimulating food should be eliminated. Coarse foods which are likely to irritate the stomach should also be withheld from diet, such as lettuce, tomatoes, cabbage, celery, and coarse bread. Foods should be confined to the soft, non-irritating type. The food should be either wholly unseasoned or seasoned very lightly. The secretion of hydrochloric acid can be depressed somewhat by the use of atropin, $\frac{1}{200}$ of a grain being given three-quarters of an hour before each meal. This quantity, however, should, at times, be increased and should usually be pushed to the point of dryness of the throat. Atropin is particularly effective in those naturally vagatonic and where the condition is a result of vagus stimulation without organic lesions such as that caused by the pulmonary reflex. When due to ulcer, appendicitis, or disease of the gall bladder, it may be of limited or no value. Its use should be persisted in for a prolonged time.

Bromides are also valuable in these cases. From 10 to 30 grains of bromide of soda may be given before meals although its action is not as certain as that of atropin.

A very important point in the treatment of hyperchlorhydria is to neutralize the acid because it not only produces discomfort in the way of a burning sensation, but, where we have large quantities, it has a tendency to produce spasm of the pylorus and interfere with the emptying of the stomach contents into the duodenum, which, if maintained long enough, will favor dilatation. It also throws a great burden upon the intestinal secretions to neutralize the excess of acid when it enters the duodenum, and thus delays the emptying of the stomach. For neutralizing the acid the old favorite combination of subcarbonate or subnitrate of bismuth, bicarbonate of soda and carbonate of magnesium, is excellent. These may be used in equal parts by weight, one teaspoonful to be given two or three hours after meals at the time when the burning begins. The bismuth has a tendency to depress the secretion of the acid, while the soda and magnesium neutralize it. If the patient is inclined to constipation the magnesium may be increased and the bismuth be decreased in amount. If, on the other hand, there is a tendency to diarrhea, the bismuth may be increased and the magnesium reduced; or, if this is not sufficient to counteract the acidity, more soda may be added. At other times plain bicarbonate of soda may be used in teaspoonful doses.

It is necessary to explain to the patient the necessity of taking sufficient of the salts to neutralize the acid; otherwise he will not understand and is apt to take small amounts and only partially neutralize and in this way obtain only partial relief. Sometimes an absolutely meat-free diet persisted in for a long time seems to help most; although, at first the acidity is worse because of the lack of albumin to combine with the acid. I have found in my work among the tuberculous, however, that a general diet is the best; and, inasmuch as I feel that some proteid is essential, I do not like to eliminate meat from the diet, and usually try to control the acidity as well as possible without eliminating it, at least, until such time as the condition of the patient will permit of more rigid diet without risk. When due to ulcer of either the stomach or duodenum, these conditions must receive appropriate treatment.

Dilatation of the Stomach.—There are many factors in tuberculosis which tend to produce dilatation of the stomach. Some of these are avoidable, others are not. In the early stages of the disease it is not apt to occur unless there is some obstruction of the pylorus. In chronic cases it formerly was often found as a result of overfeeding. It is in the late cases, however, after degenerative processes have set in and the walls have become atonic that this condition becomes a serious factor. When this condition has been attained the digestive juices are, as a rule, impaired; the muscles are weakened; motility is lessened; the food remains in the stomach longer than it should; gases develop, and the organ dilates. This often occurs during a prolonged period of toxemia when the inhibitory influences of the sympathetic nervous system are predominant. Again, it occurs in hyperacidity when the spasm of the pylorus prevents its opening naturally.

Slight degrees of dilatation may be present without troublesome symptoms arising; and a stomach when dilated to a slight degree may again become functionally normal. When, however, the severe degrees of dilatation that are encountered in advanced tuberculosis take place, in which, through toxemia, marked degeneration has become established, and delayed and imperfect digestion has resulted, the symptoms become extremely troublesome and are also difficult to relieve.

Patients suffering from dilatation of the stomach complain of loss of appetite, feeling of fulness, nausea, and vomiting. They may think they will enjoy a meal, but are usually satisfied when they have eaten a few mouthfuls. They also have a feeling of fulness on eating small amounts of food. This common symptom of dilatation—feeling of fulness—has been shown by Hertz and his co-workers[6] to be due to tension exerted on the circular muscle fibers of the stomach. Changes in the circular muscles are brought about by a reflex from the esophagus during the act of swallowing. When food passes through the esophagus it reflexly stimulates the circular muscles of the cardia to dilate and make room for the additional food which enters. If food is eaten rapidly and sufficient time is not allowed for the circular coat to dilate, a feeling of fulness results; or, if the stomach is already completely relaxed, as it is in cases of marked dilatation, then the taking of food stimulates the fibers to further relaxation, just the same as in the non-dilated organ; but, inasmuch as the circular muscular coat is already fully relaxed, further response to the esophageal swallowing reflex cannot do more than to attempt to relax an already relaxed muscle. The result, however, is the same feeling of fulness, as is noted when the organ is fully distended by food; because the circular fibers are subjected to increased tension after they are fully relaxed.

I have found it best, if this condition is severe, to feed the patient at frequent intervals; to reduce the amount of liquid taken with the meals to the minimum; and, above all else, to withhold food which the stomach digests slowly as well as such articles as produce gas. However, if the dilatation is not too severe and not causing too much disturbance a liberal diet should be insisted upon, for the condition can only hope to be improved as the general health of the patient improves. Concentrated nutritious food should make up an important part of such a dietary. Lavage is sometimes valuable in these cases. Strychnine seems to offer some aid; but building up the patient where this is possible, is of greatest importance. Unlike dilatation in the individual who is otherwise strong and healthy, we here have malnutrition and general lack of tone as a more important factor than obstruction.

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[6] The Sensibility of the Alimentary Canal, Oxford University Press, London, 1911.

Disturbance on the Part of the Intestines.—Many of the nontuberculous complications on the part of the intestinal tract are also extremely common in tuberculosis.

Enterocolitis.—This complication is a very common one. It may be of slight degree or it may be very severe. It may be due to errors in diet, or result from an obstinate constipation and the use of laxatives to which so many people are addicted; or, in tuberculosis, it is probably at times due to toxemia; and, at times, to a venous congestion in the intestinal wall, the latter being a constant factor of this disease. The excessive use of raw eggs will produce it.

According to the severity of the case these patients complain of loss of appetite, nausea, feeling of fulness and sometimes distention of the entire abdomen, accompanied quite often by colicky pains. Flatulence, general lassitude, nervous irritability and sometimes depression are common. The entire course of the colon may be sensitive to pressure. The inflammation usually involves both the large and small intestine. Constipation may be present; but, if so, it is apt to alternate with loose movements or diarrhea. The patient usually passes one or two soft stools a day and has considerable gas. These patients may usually be relieved by proper treatment although we may never be sure that we have arrived at a permanent cure, for relapses are extremely common. Treatment depends upon the severity of the case.

The most important measures in the treatment of this complication are the relief of mental depression, rest and diet. It is well for the patient to understand that the maintenance of a good result depends not on medicines but on persistently following rational living and appropriate diet. Remembering the effect of emotions on the digestive tract, the importance of optimism cannot be overestimated. If the condition is at all severe, rest in bed is essential for the proper handling of this complication as a cause, achylia must always be kept in mind.

In severe cases I put the patient to bed and eliminate all questionable foods from the diet. At first, nothing but gruel and milk or gruel and buttermilk are given, making the gruel by boiling rice or barley until it becomes a pulp, and straining this through a cloth. Water is then added to make it the consistency of thick soup. A glass of gruel and a glass of milk or buttermilk is given three or four times a day. In some instances milk will not be tolerated. Then gruels and soups may be used exclusively. In some cases this line of treatment proves wholly inadequate. Under such conditions we should recall the possibility that the colloidal boiled starch may be interfering with the peptic digestion. Sometimes it is best to eliminate starch wholly from the dietary. Cases of achylia respond to hydrochloric acid.

Colonic injections of starch water and bismuth are helpful. Starch water is made by boiling the starch and adding water so that it is of such consistency that it passes through the syringe easily. To each quart of starch water one dram of subnitrate of bismuth is added. This is used quite warm. It is injected into the rectum slowly and the patient is instructed to retain it at least for half an hour, or even longer, if possible. This is given once or twice a day at first; and, as the patient improves, the intervals are lengthened. Hot packs to the abdomen for two hours once a day or one hour twice a day are employed. As medication the patient is usually given a teaspoonful of castor oil each night. This method has proved very satisfactory in handling these patients.

Now that we fully understand the antiperistaltic movement of the colon we recognize that it is not necessary to use high flushings, as we formerly did, because fluids thrown into the rectum will soon travel the entire course of the colon; and even through the ileocecal valve if patulous. Sometimes patients will complain that they cannot take large enemas without feeling ill. They have feelings of faintness, colicky pains, and nausea owing to irritation of vagus endings.

The following diet has proved very satisfactory in the treatment of the majority of patients having enterocolitis.

Diet Permitted in Severe Cases.—At first, gruel (rice or barley), milk (diluted with half water or Vichy) or buttermilk; or gruel soups where milk is not tolerated.

As the patient improves, the following are permitted. Rice, toast, or stale bread, cereals, soups, sago, noodles, macaroni, baked or mashed potato, eggs, either raw, soft boiled, or poached.

Moderately Severe Cases.—These cases may have all of the above, and in addition such light vegetables as spinach, carrots, cauliflower, peas, and squash; and meats sparingly.

Mild Cases.—These cases may have the above with whole wheat bread, meats, fish, and fruit sauces.

In colitis it is always best to take foods warm and avoid such cold foods as ice cream, cold milk, and cold water. Hot water may be taken to advantage.

If a more laxative diet is required prunes, oatmeal, and honey may be added.

Foods Forbidden.—Coffee (especially strong), all acid or raw fruits, legumes, cheese, cabbage, smoked meat and fish, fat meat, pork, veal, fresh bread, and pastry. In cases accompanied by severe diarrhea, all extracts of meat should be withheld from the diet until acute symptoms have disappeared.

Intestinal Stasis.—The mechanical factors of digestion have assumed a much more important place in medical literature in recent years than formerly. In America this has been largely through the splendid work of W. B. Cannon.[7] We were formerly accustomed to speak only of the peristaltic action of the small intestine, but we now know that there is a definite peristaltic action of the stomach and of the large bowel. In tuberculosis stasis is extremely common. The peristaltic movements of the intestines are increased by the vagus and inhibited by the splanchnics, both of which are stimulated in tuberculosis, resulting now in spastic constipation and again in that of the atonic type. Inasmuch as congestion of the intestinal veins is present to a greater or lesser extent in all pulmonary inflammations, we have interference with the action of the peristaltic wave from this cause, even in early cases; while we have the marked toxemia and degeneration added later.

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[7] The Mechanical Factors of Digestion, Longmans Green & Co., New York, 1911; also Bodily Changes in Pain, Hunger, Fear, and Rage, D. Appleton and Co., New York, 1915.

There are several points in the alimentary tract where food is naturally hindered in its downward progress. The normal passage of food in the average sthenic individual requires about twenty-four hours in passing from the stomach to the rectum. The first point of delay is that of the pylorus, which is presided over by certain reflexes which depend primarily on the acid content of the stomach, and, secondarily, upon the neutralization of these acid contents after they pass into the duodenum. In case of an increased acidity in the stomach we have a reflex spasm of the sphincter which retards emptying and oftentimes leads to more or less dilatation of the organ. The pylorus may be kept closed reflexly by inflammations in the neighboring organs. This is particularly evident at times in cases of appendicitis, duodenal ulcer, and cholecystitis.

The next point is that of the ileocecal valve. The purpose of the closure of the ileocecal valve is probably to favor absorption. Food naturally requires about four or five hours in passing from the stomach to the ileocecal valve. When such irritations as those resulting from cholecystitis, chronic appendicitis or tuberculosis of the appendix, or inflammatory conditions about the head of the cecum, are present, the opening of the sphincter may be interfered with and stasis in the ileum result. The same is due at times, and probably more often, to mechanical obstruction. Normally, when food enters the stomach, the ileocecal valve is reflexly opened; but any irritation in the neighborhood of the valve itself may prevent this opening and favor stasis. The cecum and ascending colon is a sort of churning place for the food. Like the stomach this is another reservoir where it is retained for a considerable period of time.

The passage of food along the colon is very different from that in the small intestine. Here, instead of being pushed along by the small peristaltic waves, such as are present in the small intestine, the particles of food are carried onward more or less *en masse* by massive peristaltic waves and collect in certain portions such as the middle of the transverse colon, the pelvic colon, and the rectum.

With central stimulation of the sympathetics by toxins and other causes, with the presence of peripheral stimulation of the vagus, the venous congestion of the intestines, the degeneration in the walls of the gut, the displacements such as are common in tuberculosis, particularly after the stage of wasting has been reached, stasis in both the ileum and colon become exceedingly common and also become matters of extreme importance.

The ill effects of stasis upon the patient are mainly due to absorption of poisonous materials which generate during the slow passage of the contents in the bowel which act upon the brain centers producing general instability of function. Not only do we have the general effects of the absorption of these deleterious products to deal with, but we have a disturbance in the rhythmical function of the gastrointestinal tract. These patients suffer from toxemia, which is expressed in the symptom-complex: headache, malaise, aching more or less severe throughout various portions of the body, rapid heart action, alteration and depression of appetite, and general inhibition of the gastrointestinal functions, which results at times in marked disturbance of nutrition. Often flatulence and colicky pains are troublesome. This toxemia is often preceded by a retardation of the intestinal contents due to stimulation of the vagus. Studies in visceral neurology, as well as clinical observation, lead me to believe that, omitting cases of definite obstruction, increased tonus of the intestinal branches of the vagus is the most frequent underlying cause of intestinal stasis.

The relief of stasis is not a simple matter because patients who suffer from it, even those without tuberculosis, suffer more or less from a disturbed nerve equilibrium. It is important not only to correct the stasis for the time being, but to keep it corrected; to improve the habits of the individual; and to reeducate the nervous system. Inasmuch as this is a part of the general treatment of tuberculosis anyway, these patients should improve by the general methods which are employed in the handling of this disease.

Stasis cannot be cured by cathartics. It is important to try in every way possible to obtain the normal onward movement of the material in the gastrointestinal canal. It is very difficult to discuss this question aside from constipation, although the two are not identical. Such measures should be utilized as favor a normal passage of the food in the intestine, because by so doing the decomposition and fermentation which otherwise take place are prevented and the ill effects of absorption are avoided. Aside from this, the production of gases which are more or less harmful in these cases in that they produce dilatation and also stimulate to irregular contractions and the production of colicky pain, may be avoided. Bran, agar-agar, and paraffin oil are all valuable in the treatment of these cases. Hot applications to the abdomen have a splendid effect in relaxing the spasm which may be produced by the irritating gases. Sometimes a course of some of the mineral waters, while theoretically contraindicated, is allowable for it seems almost necessary to administer some direct relief. We are obliged to deal somewhat differently with the tuberculous, with his toxemia and degeneration, from what we do with the non-tuberculous. Such waters as Hunyadi Janos, Apenta, and many that we have in our own country, such as Pluto and Abilena, may be used. The use of these should only be temporary and withdrawn after serving their purpose. These patients should be encouraged to drink large quantities of plain water, particularly warm water. Atropin should be used freely and persistently.

Constipation.—There are many factors in tuberculosis which tend to the production of constipation or to the aggravating of such a condition when it already exists. These are:

1. The habits of previous years which have so often caused neglect of this important function of the bowels.

2. Faulty habits of eating which are more or less compensated for as long as the patient is well, but which are magnified by illness.

3. Disturbances of the normal physiologic rhythm of the gastrointestinal function, whether due to motor or sensory change or alteration in position or contour of organs.

4. The toxic and reflex influences through the sympathetic and vagus nerves and the degenerations which are essentially due to the tuberculous process.

5. The splanchnic congestion which is produced by the disturbance in the function of the respiratory muscles and the consequent reduction and limitation of the inspiratory act, as described more fully on page 301.

6. The limitation of exercise made necessary by the activity in the tuberculous focus.

7. The eating of concentrated foods to the exclusion of those of the more bulky type, partly from former habit, partly because of the misguided notion that concentrated foods are the only ones of value in tuberculosis, and partly because a disturbed appetite and repugnance for food make it difficult if not impossible to chew the food of a more bulky nature.

I have found in my own work that the classification of constipation into atonic and spastic, as suggested by Cohnheim,[8] is a very practical one from the standpoint of therapy. We find more or less constipation in a very large per cent of our cases. In some it is of the atonic type, in others of the spastic type, and more generally, a combination of the two. The treatment of these two types is radically different, consequently difficulties arise, particularly in the mixed type.

Hurst[9] also recognizes two types of constipation differing somewhat from Cohnheim. He says:

"As a result of my investigations with the x-rays on the motor functions of the intestines in health and disease, I concluded that all cases of constipation can be divided into two classes: in the first, which may be called *intestinal constipation*, the passage through the intestines is delayed, whilst defecation is normal; in the second class, for which I have adopted the term *dyschezia*, there is no delay in the arrival of the feces in the pelvic colon, but their final expulsion is not adequately performed."

Of these two types of constipation it can readily be seen that, that of the spastic type or intestinal type of Hurst is more serious than the atonic, because the contents are delayed in the passages higher up in the canal where the absorptive powers of the gut are greater and at a time when decomposition of the contents is more apt to occur. Consequently spastic or intestinal constipation is accompanied by greater toxemia. It is also accompanied by the formation of gas and sooner or later leads to destructive changes in the wall of that portion of the bowel affected. The patient suffering from atonic constipation, on the other hand, does not suffer from toxemia to so great an extent neither does he experience the deleterious effect of gas in the bowel.

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[8] Disturbances of the Digestive Canal, Translated by Fulton, J. B. Lippincott Co., 1909.
[9] The Sensibility of the Alimentary Canal, Oxford University Press, 1911.

Atonic Constipation.—Atonic constipation is that type in which the bowel itself seems to have lost its natural response to stimulation. This type of constipation is usually characterized by a large dry stool. In this form of constipation gas is not a prominent symptom, in fact it is often entirely absent. Often the only complaint of the patient is the inability to have a normal stool. Sometimes, however, they have toxic symptoms as feelings of dullness, headache, malaise, and the lack of desire for work. These patients are in the habit of relieving themselves either by the use of enemas or laxatives, both of which answer the purpose for the time, but in the end prove harmful. One measure of great importance in the treatment, particularly of this form of constipation, is somewhat difficult to carry out in tuberculosis,—that of exercise. Exercise is valuable; but we are forced in treating tuberculosis, particularly during the active stage, to keep the patient quiet. The wasting which occurs in advanced cases and the congestion of the intestinal tract have a tendency to increase constipation when present, no matter whether it be of the atonic or spastic type.

The first thing in the treatment of all forms of constipation is to get away from cathartics. In fact, they of themselves will eventually aggravate the very condition which we are trying to eliminate.

Atonic constipation requires that type of food which furnishes large amounts of waste matter. It should be rich in cellulose and of such a character as will mechanically stimulate the mucous membrane.

It is my custom to have these patients drink one or two glasses of cold water on going to bed and also the same on getting up in the morning. Fruit juices such as lemonade and orange juice may be used in the morning before breakfast to advantage. Tea and coffee should be used sparingly or not at all. Buttermilk, at least two days old, may be used. Butter should be used freely. Nearly all kinds of fruit are allowable in this form of constipation. Vegetables of the coarser varieties, such as lettuce, celery, asparagus, string beans, spinach, legumes, and tomatoes, are extremely valuable, so are coarse breads and coarse cereals. Meat and eggs produce a small amount of residue but, at the same time, should be allowed in moderation because of the fact that these patients require them in their dietary.

Any mechanical derangement of the gastrointestinal tract should be corrected if possible. Massage is of value in this form of constipation. Warm olive or linseed oil enemas given at night are effective in softening scybalous masses if present. Beginning with one ounce this may be increased to four or five ounces. The patient should retain this over night. This may be used at first every night and then the interval should be gradually lengthened until it is used only once a week. Sometimes, at first, it may be necessary to give these patients some of the laxative mineral waters, although they should be withdrawn as soon as possible. For such purposes Hunyadi Janos, Apenta, Pluto water, Carlsbad Salts, or plain phosphate of soda may be used each morning before breakfast. The addition of one or two tablespoonfuls of bran to the diet two or three times a day; or agar-agar, one teaspoonful to a tablespoonful twice a day is often of great benefit. What is desired is a normal motor response on the part of the intestinal muscles.

Hurst[10] speaks of that type of constipation which results from a delay in expelling feces from the rectum as dyschezia. This is allied to the atonic type. I will quote his discussion of the causes which operate to produce this type. It will be seen how the conditions enumerated above as being present in tuberculosis will operate in both delaying the passage through the intestine and in emptying the rectum when it has been reached. He says:

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[10] The Sensibility of the Alimentary Canal, Oxford University Press, 1911.

"The most common cause of dyschezia is the habitual disregard of the call to defecation on account either of ignorance or laziness or of fear of pain in diseases of the anus and the neighboring organs. I have already described how the sensation of fulness in the rectum passes off owing to relaxation of the tonic contraction of its muscular coat, if the call to defecation be disregarded. If it is again disregarded after its return on the arrival of more feces in the rectum, further relaxation occurs. More and more feces accumulate in the rectum, the muscular coat of which becomes more and more relaxed. As the force required to empty the rectum when overdistended with feces is much greater than that required to empty it under normal conditions, evacuation is now likely to be incomplete, even if a great effort be made. Consequently feces are constantly present in the rectum instead of only for a few minutes before defecation, and the lumen of the rectum is permanently increased owing to the atony of its muscular coat. It has sometimes been recommended that patients, who are constipated as a result of irregularity in their habits, should attempt to open their bowels after breakfast, but should not obey the call to defecation if felt at other times during the day. Our observations show that this teaching is wrong, and that, in addition to the regular morning effort a response should be made to every call, however inconvenient the time. For the occurrence of a call to defecation always means that for some reason feces have just passed from the pelvic colon into the rectum; the relaxation of tone which follows neglect of the call is undesirable, particularly in patients with dyschezia, in whom a certain degree of atony is already present.

"Dyschezia may be due to various other causes, such as weakness of the voluntary muscles of defecation and the assumption of an unsuitable position during defecation. But whatever the primary cause, the final result is the same. The incomplete evacuation of the rectum results in the accumulation of feces and in atonic dilatation.

"I believed at first that the absence in dyschezia of any sensation when the rectum contained feces was due to a blunting of the sensibility of the mucous membrane as a result of the irritation produced by the constant presence of feces. This view was shown to be erroneous by our observations that the rectal mucous membrane is normally insensitive to tactile stimulation, and that the call to defecation depends upon the sensibility of the muscular coat of the rectum. The few experiments, which I have at present had the opportunity of making, suggest that the muscle-sense is not impaired in most cases of dyschezia, as the intrarectal pressure required to produce the call of defecation is not greater than in normal individuals. The dyschezia depends upon the atonic dilatation of the rectum, an abnormally large quantity of feces being required to exert the normal adequate pressure. In extreme cases a blunting of the muscle-sense may also occur; in such cases the rapid and considerable distention produced by an enema injected with an ordinary syringe fails to produce the artificial call to defecation, which in most cases of dyschezia results in a movement of the bowels.

"There is, however, an entirely distinct class of dyschezia which depends upon deficiency of the muscle-sense of the rectum. Congenital deficiency probably causes the dyschezia, which occurs not uncommonly in infants, in whom the slight additional distention produced by the introduction of a finger or a piece of soap into the rectum results in an adequate stimulus. In the majority of cases the muscle-sense develops as the infant grows older, but congenital deficiency is occasionally the starting-point of dyschezia which lasts through life."

The place of low enemas in the relief of a loaded colon deserves some consideration. Realizing the importance of normal evacuations some faddists have gone so far as to attempt to cure all ills by injections of water into the bowel. Others, appreciating that this was a fad which was subject to abuse and capable of harm, have refused to see any good in it and have preferred to resort to the use of laxatives of various kinds. If the emptying of the lower bowel depends upon the muscle-sense, in case the muscle still possesses expulsive power, an enema should furnish the normal stimulus and be a natural measure for relief. It also has an added advantage of softening the feces.

When trying to get patients suffering from atonic constipation away from cathartics, I believe this one of the best measures. The amount of water should not be too large and is best at low temperature which stimulates the tissues while the warm relaxes them.

While the small enema of low temperature is of value in atonic constipation it is not so valuable in the spastic type because the delay in passage is due to increased tone and is farther up in the canal. This type is produced by too much muscle stimulation. Relaxing measures here are most valuable. If injections are used in this type, they should be quite warm and introduced in such a manner as to allow the water to travel upward toward the cecum. While the low enema for the relief of atonic constipation should not be held, that for spastic should be retained for several minutes.

Spastic Constipation.—Spastic constipation is very different from atonic constipation in its nature as well as in its demands for treatment. While atonic constipation is due to a failure of the bowel to respond to stimuli, spastic constipation is due to an increased vagus tonus, and an irritative condition of the intestinal tract in which the colon is the seat of spastic contraction. Spastic constipation is extremely common in cases of hyperchlorhydria. Every now and then there is an absorption of toxins with the resultant toxic syndrome. The patient feels dull, has a slight headache, and loses ambition. The bowel movements differ from those of the atonic type. They are usually of soft consistency, small, ribbon-like in appearance, and the patient experiences an unsatisfied feeling after the bowels have moved. This form of constipation is associated with flatulence. Gas is a very common factor and is often accompanied by colicky pains. Individuals who are naturally of the vagotonic type are very prone to suffer from this form of constipation. Cohnheim says that every case of chronic constipation that runs its course with attacks of pain belongs to the spastic variety, in which inflammatory and catarrhal changes of the intestinal tube are demonstrable. Patients suffering from spastic constipation are especially harmed by laxatives. In some cases laxatives even increase the constipation. I have seen cases where cathartics caused so much irritation that defecation was almost impossible.

The principles of the treatment of spastic constipation are the opposite from those for atonic constipation. In atonic constipation we endeavor to irritate and awaken in the bowel a response to stimulation; while in spastic constipation, we endeavor to allay all irritation and remove all factors which have a tendency to provoke irritation. Rest is very important in these cases. Patients do better when kept in bed for a time. Mechanical measures such as massage are contraindicated. Hot packs to the abdomen for a period of two hours daily are of great value. The oil treatment, as mentioned in atonic constipation, is of great importance; both the administration of paraffin oil by the mouth in doses of one or two ounces per day, and the administration of the oil enemas to be retained at night.

Diet is most important. Foods which produce much gas, likewise those which contain large quantities of residue, should be eliminated from the dietary. These patients should not drink cold water; but large quantities of hot water aid in relieving spasm. One or two glasses should be taken at bedtime and the same again on arising in the morning. Weak tea or coffee may be used sparingly, although the patient is better without them. Sometimes these patients may be allowed orange juice in the morning, although, at other times, I find it is apt to produce colic; and, if hyperchlorhydria is present, it is contraindicated. Milk, buttermilk (at least two days old), all soft vegetables such as peas, carrots, mashed potatoes, baked potatoes, squash, hominy (if chewed thoroughly) and cereals may be used. Vegetables are best in purees. Spinach, Brussels sprouts and cauliflower may be used at times for variety, but not often; cauliflower, particularly being prone to produce gas. Fruits such as pears, peaches, and prunes are valuable. Honey, marmalade, jellies and jams which have a laxative effect through chemical action, are very useful. Stale white bread, toast, and zweibach may be used, but hot bread, biscuits, and coarse bread are not permissible. Green vegetables must sometimes be wholly eliminated from the dietary until the symptoms have been relieved for some time.

There are two drugs which are of great value in this type of constipation, atropin and bromides. Atropin can usually be given in 1/200 grain doses three times a day, or sometimes even in doses of 1/100 grain twice or three times a day. Ten grains of bromide taken three or four times a day will often answer, though less efficaciously, the same purpose as atropin in allaying nervous irritability, and relaxing the intestinal spasm. Where gas is extremely troublesome and not relieved by atropin alone, the following prescription, as suggested by Cohnheim, has proved to be of great value in my practice.

℞	Tincturæ belladonnæ foliorum	5.0-10.0
	Spiritus menthæ piperitæ	5.0
	Tincturæ valerianæ	15.0-20.0

M.

Sig.: Thirty drops in a cup of hot carminative tea, three times a day.

Since nutrition is such an important factor in the cure of tuberculosis and in the maintaining of health, after once regained, it is essential that we should not be satisfied to use laxatives to carry our patients along during the time of treatment, but we should help them overcome their constipation when possible.

Biliousness (So-called).—While it may be undignified to treat of biliousness in a discussion of this kind, yet I deem it important to emphasize the fact that it is not an entity and that it has no place in our literature. Biliousness is supposed to be an affection of the liver, but, in reality is an acute disturbance in the gastrointestinal tract accompanied by toxemia. It is a group of symptoms,—those of headache, feeling of malaise, lack of desire for work, mental hebetude, sometimes nervousness, variable or lack of appetite, coated tongue, and constipation. This might be due to alterations in the gastric or intestinal secretions, to disturbances in motility, or both; or it might be due to some derangement of the important glands associated with digestion. I believe the greatest factor is an acute stasis of bowel contents with absorption of toxins. Inasmuch as this condition is usually associated with acute constipation, it is treated with laxatives. In my work I have gradually come to rely particularly on one laxative, and that is, castor oil. My instructions to patients when suffering from this chain of symptoms is to stop all food or reduce it to a minimum for a day, and take a dose of castor oil. While there is much prejudice against castor oil, I find in my own practice that I am gradually limiting myself more and more to its use. It seems to be the most efficacious of any remedy that I have used. It can be made quite pleasant if given in orange juice. Put the juice of half an orange in the bottom of a small medicine glass. Then put in from three-quarters of an ounce to an ounce of oil and cover it over with the juice of the other half of the orange. Let the patient swallow it quickly. It may also be taken in coffee, but I prefer the orange juice. Lemon juice can take the place of orange juice, although it is not quite so pleasant to take. Where there is great objection to castor oil, saturated solution of epsom salts in half ounce doses may be used, or a teaspoonful of saturated solution may be given every hour until a thorough movement of the bowels has been obtained. A compound cathartic pill, at night, is also quite valuable. Calomel may be used to advantage. It may be used in small doses of one-tenth grain every hour until a grain has been given, followed either by castor oil or epsom salts; or, two grains may be given at night, a half grain every half hour until the quantity is taken, to be followed by oil or salts the following morning. The griping caused by calomel may be prevented by the use of some form of hyoscyamus or atropin. In my practice I rarely use any other remedy than castor oil or an enema. These are non-irritating and seem to be the least harmful.

Nervous Influences in Gastrointestinal Disturbances.—While errors in diet are many and pathological changes in the gastrointestinal tract not infrequent, yet it is well for both physician and patient to learn that digestive disturbances are just as likely, in fact, more likely to come from within the patient himself and be partially or wholly within his control. Emotional influences, such as those produced by pain, anger, fear and pessimism are exceedingly apt to disturb the gastrointestinal function. While we are constantly looking for mechanical and secretory disturbances in the intestinal tracts of our tuberculous patients we must not forget the very important influence which emotion bears to them and to nutrition in general. If we can keep the patient happy, cheerful, optimistic, hopeful, and free from pain, his digestion is better, he sleeps better, and all organs functionate better. On the other hand, if the patient is pessimistic, worrying about every little thing that comes up and allowing himself to be depressed and discouraged by trifles, he is apt to have many attacks of indigestion which he could escape by self-control.

There is a great deal in the saying, "Laugh and grow fat," because with happiness comes better digestion and better assimilation.

The effect of emotion on peristalsis is shown by Cannon.[11] He says:

"In my earliest observations on the stomach I had difficulty, because in some animals peristalsis was perfectly evident, in others there was no sign of activity. Several weeks passed before I discovered that this difference in response to the presence of food in the stomach was associated with the difference of sex. The male cats were restive and excited on being fastened to the holder, and under these circumstances gastric peristalsis was absent; the female cats, especially if elderly, submitted with calmness to the restraint, and in them peristaltic waves took their normal course. Once a female with kittens turned from her state of quiet contentment to one of apparent restless anxiety. The movements of the stomach immediately stopped and only started again after the animal had been petted and began to purr. I later found that by covering the cat's mouth and nose with the fingers until a slight distress of breathing occurred the stomach movements could be stopped at will. Thus, in the cat, any sign of rage, or distress, or mere anxiety, was accompanied by a total cessation of the movements of the stomach. I have watched with the x-rays the stomach of the male cat for more than an hour, during which time there was not the slightest beginning of peristaltic activity, and yet the only visible indication of excitement in the animal was a continued to-and-fro twitching of the tail.

"What is true of the cat has been proved true also of the rabbit, dog, and guinea pig. A female cat that ordinarily lies quietly in the holder, and makes no demonstration will occasionally, with only a little premonitory restlessness, suddenly fly into a rage, lashing her tail from side to side, pulling and jerking with every limb, and biting at everything near her head. During such excitement and for some moments after the animal has become pacified again, the movements of both the large and small intestines entirely cease. The opposing influences, reaching the alimentary canal by way of the sympathetic system during emotional excitement, can wholly destroy both the secretory and motor activities which have been started by the bulbar system. The importance of avoiding so far as possible the states of worry and anxiety, and of not permitting grief and anger and other violent emotions, to prevail unduly, is not commonly appreciated; for the subtle alterations wrought by these emotional disturbances are uncommon to consciousness and have become clearly demonstrated solely through physiological studies. Only as the consequences of mental states favorable and unfavorable to normal digestion are better understood can good results be sought and bad results avoided, or, if not avoided, regarded and treated with intelligence."

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[11] The Mechanical Factors of Digestion, Longmans, Green & Co., New York, 1911.

Amyloid Degeneration.—In advanced tuberculosis the vessels of the intestinal tract often become the seat of amyloid degeneration. This causes the interference with secretion, motility, and absorption, and is especially serious because it cannot be remedied.

Errors in Diet.—Errors in diet should be avoided as much as possible where a high state of nutrition is desirable. Errors in diet may be due to the eating of the wrong kind of food, or to eating too much or too little food, or to eating food which is not suited to the nutritive requirements of the patient or the peculiar digestive conditions present. In this connection it must be borne in mind that the worrying, fretting, pessimistic patient is just as apt to disagree with his food as his food is to disagree with him; and that he often worries about an indigestion that he alone can prevent.

CHAPTER XI.

COMPENSATORY CHANGES IN THE THORACIC AND ABDOMINAL CAVITIES RESULTING FROM PULMONARY TUBERCULOSIS.

When we consider the compensatory changes which take place in pulmonary tuberculosis, particularly in the advanced disease, we are forced to see clearly that while this is primarily a disease of the lung, it is accompanied by secondary changes which affect every organ in the thoracic and abdominal cavities.

These compensatory changes must be discussed together. They are so intimately related that it is impossible to completely separate those which take place between the organs within the chest itself from those which take place between the organs of the abdominal and thoracic cavities. This can be understood by considering the thoracic and abdominal cavities as one large cavity with a flexible partition separating them. No increase of tissue or loss of tissue can occur in one part of this cavity without affecting all parts; neither can any increase or decrease in the contour of the walls take place in any one portion of this cavity without affecting other or all portions.

The symptoms which are produced as a result of the disturbed function of the organs and parts which take part in the production of compensation are such as might be caused by other conditions present as well; consequently, the reader must bear in mind that while the symptoms mentioned may be caused by the conditions under discussion, they are not, necessarily, wholly due to them. It is often impossible to assign the true cause to each symptom because several conditions all of which are present, are capable of producing it. So, in reading this chapter, let it be borne in mind that the symptoms here mentioned may be caused by many conditions, among which are the shifting of organs and resulting compensatory changes described.

Compensatory Changes Taking Place Within the Thoracic Cavity.—In order to understand the extent of compensatory change which takes place between the organs within the thorax in pulmonary tuberculosis, it is necessary to consider the thoracic cavity as a cone-shaped cavity whose walls on all sides, except the base which is a thin flexible membrane, are made up of a framework of bones. All of these walls, even the bony ones, are somewhat yielding to pressure but they maintain their position and shape under conditions of health, and, as a rule, change their contour only when the organs within, or adjacent, are affected by pathological changes. Changes in the contour of the flexible membranous wall (diaphragm) occur and will be discussed fully later on; but it is necessary to state at this time that the position of the diaphragm is a peculiarly sensitive one. It depends upon the relative pressures in the thoracic and abdominal cavities. Normally, the pressure in the thoracic cavity is negative while that of the abdominal cavity is positive. The negative pressure of the thoracic cavity tends to draw or suck the diaphragm upward, while the positive pressure of the abdominal cavity tends to push it upward and hold it there.

The thoracic cavity is divided by the structures of the mediastinum into a right and left portion. These dividing structures are movable. They shift from one side to the other as conditions demand. They respond to any increase and decrease of substance or pressure in either the lung or pleura by shifting their position. In this connection the compensatory character of lung tissue must also be considered. The lung is subject to many pathological conditions in some of which there is a loss of tissue either confined to some small area or affecting a large area; in others the air cells dilate and the lung enlarges, either in circumscribed areas or generally. As a rule, the loss of tissue is compensated for, as far as possible, by enlargement of other portions of the lung or lungs (compensatory emphysema).

These compensatory changes are unavoidable; in fact, they are very desirable under the circumstances; yet they are abnormal conditions which are, according to their extent, productive of symptoms of greater or lesser severity.

To appreciate the compensatory changes which take place between the two sides of the thorax a correct conception of the mediastinum must be had. The mediastinum may be likened to a swinging door in the center of the thoracic cavity. It swings back and forth to either side as necessity demands. It is hinged to the posterior wall by the arteries and veins which pass between the large blood vessels and the intercostal structures. The anterior portion is fastened to the under surface of the sternum by the pericardium,—a structure which varies in size, it being usually large, and, in some instances, excessively large, permits of considerable freedom in the motion of the anterior portion of the door (to carry out the figure); consequently a contraction of the tissues on one side, with compensatory enlargement on the other, permits the door to swing toward the side of contraction. The extent of its motion is limited only by the size of the pericardium and the amount of play that it will permit.

In discussing this subject in a former paper,[1] the writer said: "It is somewhat surprising that these conditions have not received more attention from clinicians when we consider the fact that they are present and produce symptoms in nearly all patients who are suffering from tuberculosis. There are displacements of the heart in some form and to some degree in practically all cases where either a lessening or increase in the volume of the lung occurs. The function of the diaphragm is interfered with from the very beginning of tuberculosis. It is displaced either upwards or downwards in nearly all patients who go on to an advanced stage of the disease, the dislocation depending upon the relationship between the intra-abdominal and intrathoracic pressures.

"The symptoms which result from these conditions can only be understood in connection with the physiology of the heart and diaphragm. The heart is normally placed in the thoracic cavity in a position which enables it to perform its function with ease. The circulation of the blood is aided by every normal respiration. Any condition which changes the position of the heart, putting it in a position less favorable to action, and any condition which interferes with the full and free respiratory movements, therefore, interferes with the circulation of the blood.''

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[1] Displacements of the Heart and Diaphragm Together With Disturbances in the Function of the Latter as Causes of Symptoms in Pulmonary Tuberculosis, Interstate Medical Journal, vol. xviii, No. 6, 1911.

Aside from the symptoms due to abnormal conditions in the heart and diaphragm there are many which are due to the changes in the lung.

When ulceration, fibrosis, or both combined, are sufficiently extensive to call for a compensatory emphysema, the respiratory function is more or less interfered with and circulatory disturbances make their appearance.

The thorax is an open cavity in which a positive pressure exists, and at every physiological enlargement of this cavity, such as occurs during inspiration, the air in the bronchi and air cells attempts with all the pressure of the atmosphere back of it, to fill in the enlarged space. When there is a pathological diminution of pulmonary tissue such as occurs in tuberculosis when destruction takes place, the air in the remaining portions of the lung attempts to keep the thoracic cavity full. The result in these pathological conditions where the pulmonary tissue has wasted to any extent, is that the thin-walled air cells of the remaining portion of the lung yield to the atmospheric pressure, dilate and produce an enlargement of that portion of the lung involved (compensatory emphysema). .

An equilibrium between the intrathoracic and intra-abdominal pressures on the one hand and between these and the atmospheric pressure on the other hand, both as it surrounds the surface of the body and as it fills the air chambers of the lung, must be maintained under all circumstances if the proper functionating capacity of the various organs in these cavities is to be maintained.

To the extent to which pulmonary tuberculosis disturbs this equilibrium without full compensation; and to the extent to which the compensatory changes which occur, take place to the disadvantage of the various functionating powers of the organs concerned, is the disease responsible for the pathological changes and untoward symptoms.

Of the many pathological changes which take place in the lungs and pleura, some cause so little change in tissue that their effect on the mediastinum and diaphragm is negligible; but any considerable loss of lung tissue, or any marked decrease in the respiratory function in one part of the lung, is followed by a compensatory increase in other parts.

Man is naturally endowed with an excess of lung tissue over what is actually needed for the ordinary acts of life. This enables him to respond to unusual effort and strain by calling his reserve lung power into action. It has been estimated that a man can exist on about one-twelfth of his lung area; be fairly active on one-half and perform most of the ordinary acts of life without strain, on two-thirds. A person who has lost one-third of his pulmonary area, however, would not be able to measure up to any marked sudden effort or strain, for he has no reserve lung area to call into action.

In nature's attempts to compensate for loss of tissue, she sometimes defeats her purpose by producing conditions which by themselves are deleterious. Thus, at times, such a degree of compensatory emphysema is produced in advanced destructive tuberculous lesions that this of itself proves to be harmful to the patient, by the reduced functionating capacity of the lung tissue involved, and by the extra burden thrown on the heart as a result of it. When an extensive degree of emphysema is present the heart is less able to cope with the extra work too, because it has been subjected to more or less degenerative change owing to the destructive process in the lung and the general malnutrition and because it is forced out of its natural position to the right or to the left and is working at a disadvantage on this account.

Shifting of Mediastinum.—The shifting of the mediastinum to a recognizable degree is extremely common in advanced tuberculosis. The amount of this shifting depends on a number of factors. The mediastinum, as a whole, shifts to the left easier than to the right. The anterior mediastinum shifts easier than the posterior because the aorta in the posterior mediastinum is held fairly firmly in its position by the intercostal branches which it gives off to pass into the structures on either side of the vertebral column. The large vessels are also fixed to a certain extent by the large branches which pass up into the neck. The heart, on the other hand, which fills the anterior mediastinum in its lower portion, is contained in the pericardium, a sac varying much in size, and allowing of considerable movement on the part of the heart lying within. The amount of movement which the heart can make in health and the ease as well as the extent to which the heart moves under pathological conditions depends largely upon the size of the pericardium. While the structures in the upper portion of the mediastinum do not shift their position readily, yet they offer so little resistance that the enlarged lung on the side opposite the destruction may gradually force its way between it and the anterior surface of the chest wall over to the other side of the median line. In one of my cases (Fig. 40) the left lung had formed a new lobe which had forced its way three inches beyond the median line to the right.

The pericardium is attached anteriorly to the under surface of the sternum, inferiorly to the diaphragm, superiorly to the great vessels, and laterally to the right and left pleura.

In advanced tuberculosis, the inferior and two lateral attachments are unstable and subject to considerable change in position, thus allowing the heart to shift its position.

Displacement of the Heart.—In my practice I have found severe destructive changes of wide extent to be most common in the left lung, the upper lobe being involved, as a rule, more extensively than the lower; although large cavities and extensive fibrosis are often formed in the lower lobe also. The right lung is severely diseased less often; but when it is, the conditions duplicate those in the left, the upper portion showing the more extensive process. That the upper lobe should be the seat of the more extensive involvement is evident from the fact that the primary pulmonary infection is usually near the apex whether it be the first apex involved or the extension to the other lung; and, the disease spreads downward contiguously from the original metastasis. From this fact it is also evident that the greatest evidence of healing with the formation of fibrous tissue should be found in the upper lobes where the lesion is oldest and where the process was present before the patient's recuperative powers were so severely taxed. While we often find extensive healing in the lower lobes, yet, being, as a rule, a later infection, heal-

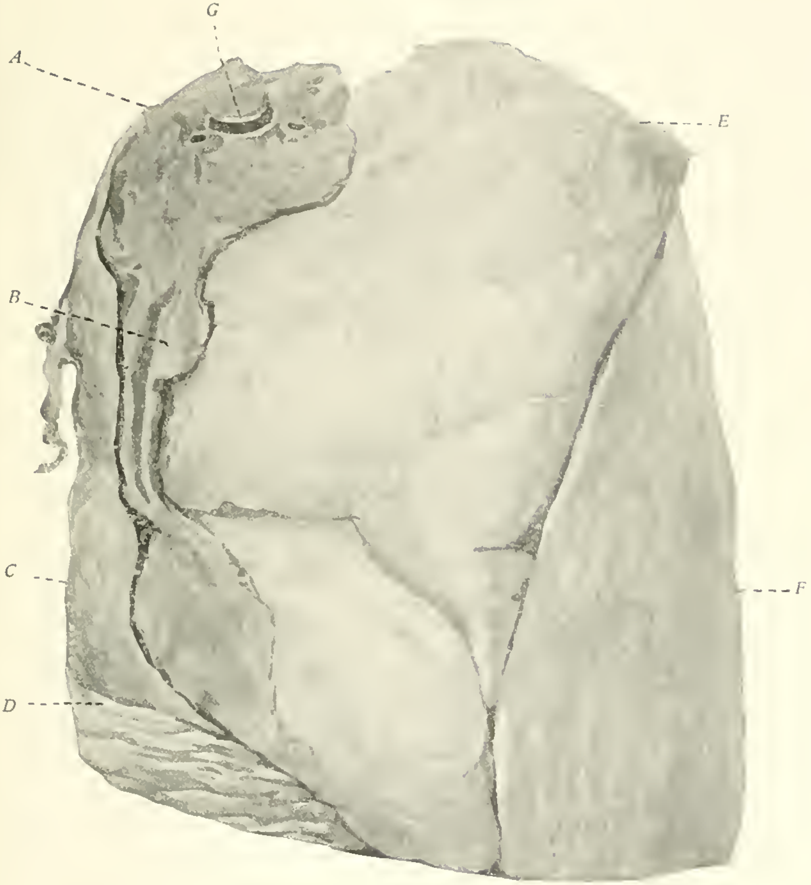

Fig. 40.—Illustrating the distortion of the thoracic viscera in a patient with a marked destructive lesion in the right lung, and marked compensatory changes in the left. *A*, the upper lobe on the right, is a small fibroid mass; *B*, the middle lobe, is only a fibrous string; *C*, the lower lobe, barely presents anteriorly, but posteriorly, was the seat of emphysema; *D*, a portion of the diaphragm, above and to the left of which lies the heart, three-fourths of which lies to the right of the median line; *E*, the upper lobe on the left represents a large portion of the lung which presents anteriorly. A new lobe has been formed, pushing through the anterior mediastinum to a distance of three inches beyond the median line; *F*, the lower lobe is also markedly emphysematous; *G*, trachea.

ing does not take place as extensively here as in the upper lobes.

The direct factors which cause the heart to change its position are the loss of lung tissue and coincident compensatory enlargement of other portions. In the final analysis, however, it is due to the necessity of maintaining a definite pressure equilibrium between the air containing chambers within the lung and the atmospheric air surrounding the outer chest wall. The bony cage yields some, but cannot make up for the entire deficiency; neither would we expect it to yield except after all more pliable structures had yielded to their full extent, unless the contraction of the muscles was a factor. The diaphragm pushes upward, but this too is limited in its ability to satisfy the demand, so it must be met by the tissues within the thorax itself. The pleural sac remains closed because of its negative pressure, or it may be obliterated because of adhesions; consequently the only method of meeting the demand which cannot be met by the diminishing of the size of the bony cage, and by the ascent of the diaphragm, is by enlarging (compensatory emphysema) that portion of the lung which is not destroyed by ulceration and fibrosis.

Such changes disturb the nicely balanced symmetry which exists normally between the two sides of the thoracic cavity and between it and the abdominal cavity and cause a distortion of all the structures within the thoracic cage. The amount of distortion is determined by the amount of destruction in the pulmonary tissue, the degree of compensatory emphysema, and the size of the pericardium and the amount of displacement which it will permit the heart to undergo.

The entire mediastinum is disturbed in its relationship but the heart is the organ that shows the greatest displacement. These displacements vary according to the character of the changes in the lung. In this present discussion I am omitting consideration of those acute displacements which occur as a result of changes in the pleura.

I have noted the following relationship between displacements of the heart and the pulmonary involvement.

1. If there is a small lesion in one or both lungs, there is no appreciable change in the position of the heart.

2. If the lesion becomes more extensive and heals there is a

tendency to draw the borders of the lung upward, and with them the pericardium. This is counteracted, however, as a rule, by a compensatory emphysema developing which affects the lower portion of the lungs and holds the diaphragm somewhere near its normal position or even pushes it lower. I have seen a few hearts that seemed to be raised *in toto* because of such a double lesion. The effect of such a change in position is to shorten the distance between the apex and the large vessels, thus producing a tendency to pouch the aorta.

3. At times, when a double involvement exists at the apices, the opposite occurs. A high grade of compensatory emphysema develops pushing the diaphragm downward. The heart follows the diaphragm and hangs suspended from the great vessels the same as it does in those enteroptotic individuals who are occasionally seen who suffer from *suspensio cordis*. I have seen several cases such as these. When examined by the fluoroscope during inspiration, as the diaphragm sinks, the heart, being unable to follow, hangs suspended, dragging on the aorta. Such is the case in many patients of the asthenic type.

4. The most common extensive displacement as mentioned above, is the one when the heart is displaced to the left and upwards (Fig. 41). In advanced tuberculosis a degenerative process which goes on to the destruction of the greater portion of the upper left lobe is not uncommon. A portion of the lower lobe is usually destroyed at the same time. Such a condition may occur and yet, the patient, if properly treated, obtain an arrestment of his disease and regain fairly good health. If an arrestment takes place it is accompanied by the formation of fibrous tissue and contraction. The walls of the cavity or cavities are compressed and the lung area which was involved in the severe destructive process is greatly reduced in size. At times a very marked contraction occurs, even though it was not preceded by a destructive process with cavity formation, the process from the start being one accompanied by the formation of fibrous tissue. Under such circumstances compensation takes place, not only in the lower portions of the left lung, but also in the right lung. Equilibrium can only be established when the space in the thorax occupied by that portion of the destroyed and con-

tracted left lung is occupied by other tissue. To this end, the right lung enlarges under the pressure of the atmosphere and gradually pushes over against the mediastinum, forcing the heart, which is also being pulled by the contracting lung, over toward the left. The diaphragm pushes up at the same time from below and this forces the apex of the displaced heart upwards, the two forces together pushing it to the left and upward. This displacement is often so great that the entire heart is to the left of the sternum.

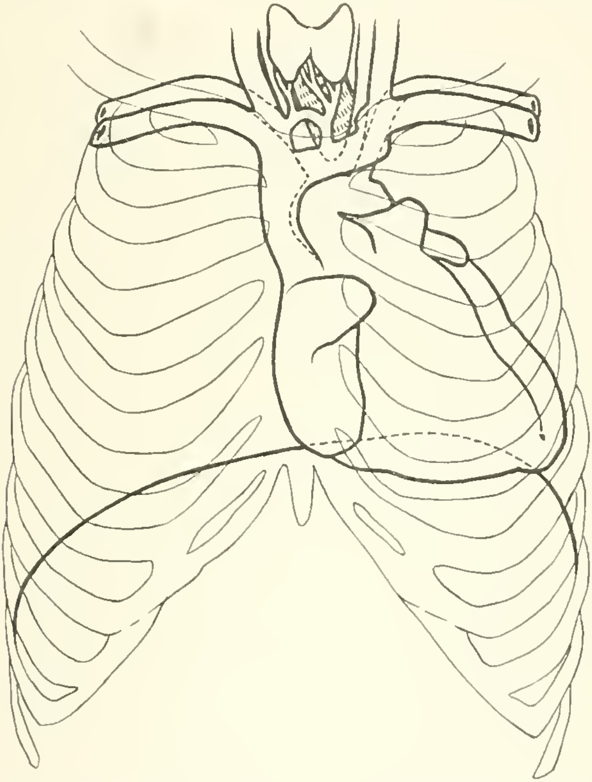

Fig. 41.—Illustrating schematically the displacement of the heart to the left. It will be seen that the left half of the diaphragm is pushed upward, and the apex of the heart follows the fifth interspace. This displacement has a tendency to reduce the curve in the arch of the aorta. The trachea may be drawn entirely to the left of the median line as shown in the cut.

This displacement not only causes the heart to be thrown out of its normal position and work at a disadvantage because of this; but the pericardium still maintaining its attachments to the sternum and center of the diaphragm is unquestionably pushed so far from its moorings that its surfaces are brought into contact in such a manner that it materially reduces the pericardial space.

This altered position of the heart causes marked dragging on the great vessels at its base, decreasing the curve of the arch of the aorta, thus bringing about a condition which interferes with the free flow of blood into the systemic arteries.

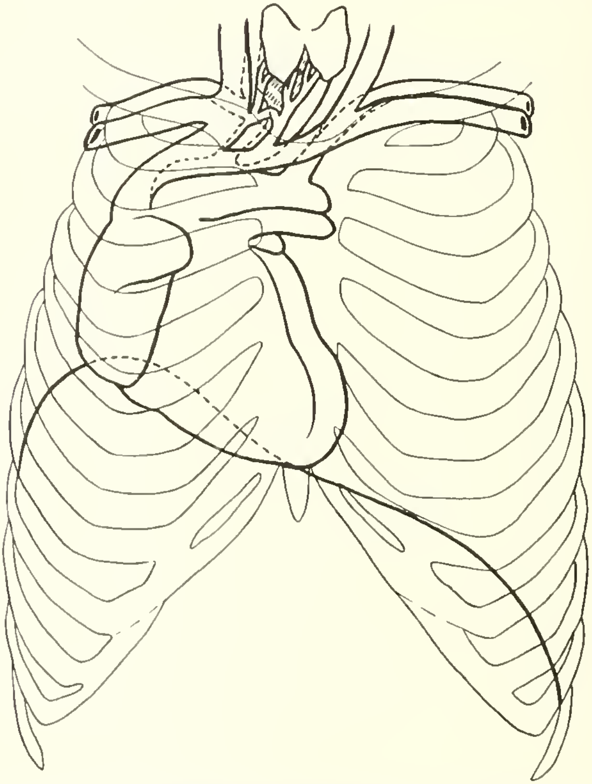

Fig. 42.—Illustrating schematically marked displacement of the heart to the right. It will be noticed that the heart is pushed upward and over. The right side of the diaphragm assumes a high position; while the left side assumes a low one in order to accommodate the left lung which is the seat of compensatory emphysema. When this displacement is present, the curve in the arch of the aorta is less acute with a tendency to pouching.

The same dragging force exerts a pull upon the other vessels at the base of the heart, drawing the pulmonary artery and large veins out of their normal course and interfering with the outflow of blood from the right ventricle and the return flow to the auricles. Dragging on the trachea often provokes cough.

5. If the severe destruction occurs in the right lung, instead of the left, the heart is displaced upwards and to the right as shown in Fig. 42. When the destruction and contraction are very marked, the heart may be displaced entirely to the right of the sternum, but this is rare compared with the total displacement to the left.

This displacement is not as natural as that toward the left. The heart lies on the diaphragm in such a way that it can be pushed to the left with ease, but, when pushed to the right, its apex meets the obstruction of the central portion of the diaphragm and the underlying liver. And, too, the diaphragm and liver are pushing up into the thoracic space at the same time, in order to help compensate for the loss of tissue. The result is that the heart as a whole is lifted as well as pushed over to the right.

The effect of such a new position is to push up and widen the arch of the aorta. This creates a relaxed condition, with a tendency to pouching.

The distortion of the other vessels is also marked and, the free flow of blood to and from the heart is impeded.

The same constriction of the pericardial space ensues as above described and the heart is forced to work in a position even more unfavorable than that mentioned above.

Not only is it more difficult for this displacement to take place, but in my experience, it proves more embarrassing to the heart which gives out more quickly under it.

As a result of these marked compensatory displacements in the thorax, the trachea is drawn or pushed over toward the side which is the seat of the destructive process. This can usually be determined by noting the position of the larynx and following the direction of the trachea as it passes down to enter the thorax. It is a suggestive point in the diagnosis of this condition.

In these displacements the recurrent laryngeal nerve is often disturbed, a brassy cough resulting similar to that which is often described in aneurysm. At times there is also noted a marked difference in the blood pressure in the two arms which might also add to the suspicion of aneurism.

Effect of Displacements of Heart.—To understand the full significance of these displacements and their effect upon the heart, we must discuss other factors than those just mentioned which have a tendency to embarrass the heart's action. Among such are the pleural adhesions which are nearly always present; the displaced diaphragm with its consequent disturbed function which will be discussed later in this chapter; the resistance offered in the pulmonary circulation by the destruction of vessels in the contracted areas as well as the embarrassment imposed by the emphysematous areas; the thickening of the arteries which results from the tuberculous infection,[2] and the degenerations of the heart muscle which result from the action of the specific bacillary and other toxins as well as the malnutrition present.

The heart is called upon to carry an enormously increased load; at the same time its muscle is weakened, and it is forced to work under disadvantageous conditions.

The cause of death in these cases of severe destruction and marked displacement is nearly always degeneration and dilatation. The patients live as long as their hearts will permit them to live.

The symptoms which occur during the time that the heart is changing its position depend on the rapidity with which such change takes place. In some cases we see the displacement take place rapidly as in the case mentioned in one of my former papers,[3] but, as a rule, several months are occupied in shifting the position. Since the diaphragm is disturbed in its function and since there is evidence of activity in the pulmonary tissue at the same time, it is extremely difficult to separate the symptoms which belong to the one condition from those that belong to the other. From the nature of the case they would be those which belong to the group of cardioneurotic symptoms such as weakness, lack of endurance, slight dyspnea, dizziness, faintness, disturbed appetite, nausea, and increased pulse rate. When these changes are occurring the patient can be kept comfortable by remaining at rest as all of the symptoms are increased on exertion. Low blood pressure is probably an important etiological factor in this chain of symptoms.

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[2] Pottenger: The Effect of Tuberculosis on the Heart, Archives of Internal Medicine, vol. iv, 1909.

[3] Displacements of the Heart and Diaphragm Together With Disturbances in the Function of the Latter as Causes of Symptoms in Pulmonary Tuberculosis, Interstate Medical Journal, 1911, vol. xviii, no. 6.

Displacements of the mediastinum should be looked for in every case of advanced tuberculosis which has suffered the loss of tissue to any extent. The condition will often suggest itself to the alert examiner when he inspects the chest, for one side with its narrow interspaces will be small, suggesting contraction, and the other large and bulging with wide intercostal spaces, suggesting emphysema. The heart impulse is often visible and shows either to the right or left of the normal place for the apex. This impulse must not be taken as the apex. Very often it is produced by the right ventricle instead. Palpation or percussion will usually show the deep borders of the heart displaced and examination of the larynx and trachea will show that they are drawing toward the side of contraction as mentioned above. The second pulmonary sound is usually markedly accentuated because of the increased intrapulmonary pressure and also because the valve is uncovered when the contraction is on the left. The second aortic sound is also often increased. But, on the other hand, the sounds may be weaker than normal when the shifting is occurring, owing to the weakened condition of the heart muscle at the time.

Compensatory Changes in Thoracic Cage.—A discussion of this subject would not be complete without mentioning the part taken in compensatory changes by the bony framework of the thorax. Changes in contour of the thoracic cage are extremely common. Contraction occurs in one portion, expansion in another, and flattening is sometimes more or less general. All of these changes must be looked upon as a part of the one ever present scheme of maintaining an equilibrium between the air pressure without the body and that within the air chambers of the lungs.

If the contractions which take place in the bony cage are purely passive in character, then we would not expect them to occur except as a last resort, after the structures within the thorax and abdominal cavities had been unable to make up for the tissue lost. Given a movable mediastinum which presumes a large non-adherent pericardium, permitting of easy and wide movement of the heart; a lung fairly free from disease on the other side, capable of taking upon itself a high degree of compensatory emphysema; and a diaphragm capable of moving, we would not expect any passive contractions of the chest wall until these soft movable tissues had exhausted their capabilities of compensation, or, in other words, until the force necessary to compensate affected the bony framework rather than the usually more movable soft tissues.

It must be borne in mind, however, that the action of the ribs on the affected side is not normal, not from the time the early infection of the pulmonary parenchyma takes place. There is a shortening of the respiratory muscles which are reflexly thrown into contraction. There is a decrease in the elasticity of the lung tissue, and a general limiting of the inspiratory act due to the fact that many of the respiratory muscles, including the diaphragm and scaleni, have the inspiratory function reflexly disturbed. As a result, the side of the chest affected, does not take its normal part in the inspiratory act and it becomes relatively somewhat smaller than the other side.

It seems but natural that the contraction of the muscles, which exists for a prolonged time, should exert an active force in producing a certain amount of compression of the bony cage.[4] Study of the mechanics of respiration emphasizes this.

When we see local contractions in one portion of the bony cage and enlargements in another, particularly when the contraction is confined to the ribs over one lung and the enlargement to those over the other, we must assume that certain obstacles have been met, and certain difficulties in establishing equilibrium by means of the intrathoracic and intra-abdominal structures have presented themselves which could not be overcome, and that the bony cage has been forced into the compensatory scheme as being the structure most yielding at this time.

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[4] Pottenger: Muscle Spasm and Degeneration in Intrathoracic Inflammations and Light Touch Palpation, C. V. Mosby Company, St. Louis, 1912.

Such a condition aside from the changes which result from muscular contractions, to my mind, could only exist when all compensation possible had taken place between the thorax and abdominal cavities and when there was an obstruction to the further shifting of the mediastinum. Therefore, it would seem probable that in such cases the heart had pushed over as far as its pericardium would allow it to push; and that the enlarged lung was still being enlarged by the atmospheric pressure, and not being able to move further toward its fellow, and likewise meeting obstacles from below the diaphragm, expended its crowding force against the ribs which at the time offered the least resistance. The principle that all bodies move in the direction of least resistance applies here, so we cannot expect an enlargement of the bony thorax until such a time as this enlargement takes place easier than enlargement in other directions.

In speaking of the bony cage being resistant, this must be understood as being in a relative sense only. We recognize that the contraction and enlargement that occurs takes place by depressing and lifting the ribs on their articulations, by changing the angle of articulation.

COMPENSATORY CHANGES TAKING PLACE BETWEEN THE THORACIC AND ABDOMINAL CAVITIES IN PULMONARY TUBERCULOSIS.

This subject has received but scant attention as yet in medical literature; but it is one that demands more consideration, if we are to understand the circulatory changes which affect the tuberculous patient in any stage of the disease, but particularly the one suffering from the advanced stage.

As the compensatory changes in the thoracic cavity show in displacements of the mediastinum, particularly the heart, so the compensatory changes between the thoracic and abdominal cavities show in displacements of the diaphragm.

The Inspiratory Act.—In order to understand the displacements of the diaphragm it is important to study the function of this important muscle and its relationship to the other muscles of respiration. I would refer those who may be interested, to the writings of Hasse,[5] Keith,[6] Wenckebach,[7] and Eppinger.[8]

The diaphragm must be looked upon as being the chief muscle of respiration and, as such, it has a very pronounced effect upon the circulation; for the flow of blood toward the heart, and indirectly the entire circulation, is greatly influenced by the phases of respiration. Its position differs according to the age of the patient, as shown in Figs. 54, 55, and 56, Chapter XIII.

Inspiration is a muscular act and takes place through contraction and sinking of the central tendon of the diaphragm innervated by the phrenics and a simultaneous lifting of the ribs. The lifting of the ribs, in quiet breathing, is brought about by contraction of the intercostales externi, the intercostales interni and the intercartilaginei, supplied by the intercostal nerves and the levatores costarum supplied by branches from the dorsal nerves.

The lifting of the ribs in forced inspiration is aided by the accessory muscles of respiration. These are:

1. The three scaleni innervated by branches from the cervical and brachial plexuses.

2. Serratus posticus superior, innervated by the dorsalis scapulæ from the fifth cervical.

3. Sternocleidomastoideus innervated by the accessorius and branches from the second and third cervical nerves.

4. Trapezius innervated by the accessorius and branches from the third and fourth cervical nerves.

5. Rhomboidei innervated by the dorsalis scapulæ.

6. The extensores columnæ vertebrales innervated by the posterior branches of the spinal nerves.

7. Pectoralis minor innervated by branches from the anterior thoracic nerves.

Conditions which interfere with the action of the diaphragm or the first group of muscles mentioned will interfere with the normal quiet respiratory act; likewise conditions which interfere with the diaphragm and the second group of muscles mentioned will interfere with forced respiration. Increased tone (spasm) or degeneration of these various muscles, by which their action is altered, such as is present in tuberculosis, or displacement of the diaphragm which is so common in advanced tuber-

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[5] Die Atmung und der venose Blutstrom, Archiv für Anatomie und Physiologie, Abt., 1906.

[6] Further Advances in Physiology, Hill, London, 1909.

[7] Über pathologische Beziehungen zwischen Atmung und Krieislauf beim Menchen, Sammlung Klinischer Vorträge (Volkmann) Innere Medizin, No. 140 and 141, 1907.

[8] Allgemeine und Spezielle Pathologie des Zwerchfells, Alfred Holder, Wien und Leipzig, 1911.

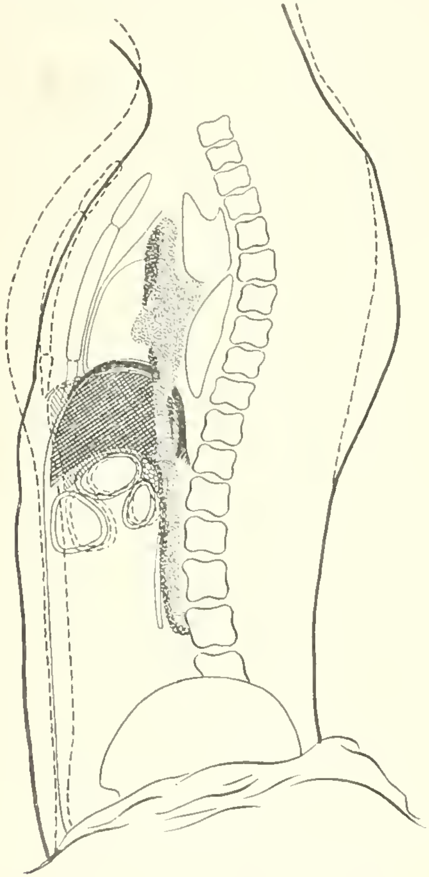

Fig. 43.—Showing the movements of the diaphragm and thoracic and abdominal walls as well as the change in position of the intrathoracic and intra-abdominal viscera during respiration of the thoracic type. The movements are from the solid lines on expiration to the broken lines on inspiration. (Hasse.)

culosis interferes with inspiration and indirectly exercises a deleterious effect upon the flow of blood to and from the heart. The influence of respiration upon the circulation may be understood by studying the changes which occur in the thoracic and abdominal cavities during the act of breathing. These can be seen by studying the accompanying figures after Hasse. Fig. 43 illustrates normal breathing of the thoracic type. The movements of the various structures and organs are from the position of the solid lines on expiration to that of the broken lines on inspiration. The entire chest wall anteriorly and posteriorly is carried forward, the diaphragm is shortened in its anteroposterior diameter and the sternum is raised and pushed forward. Fig.

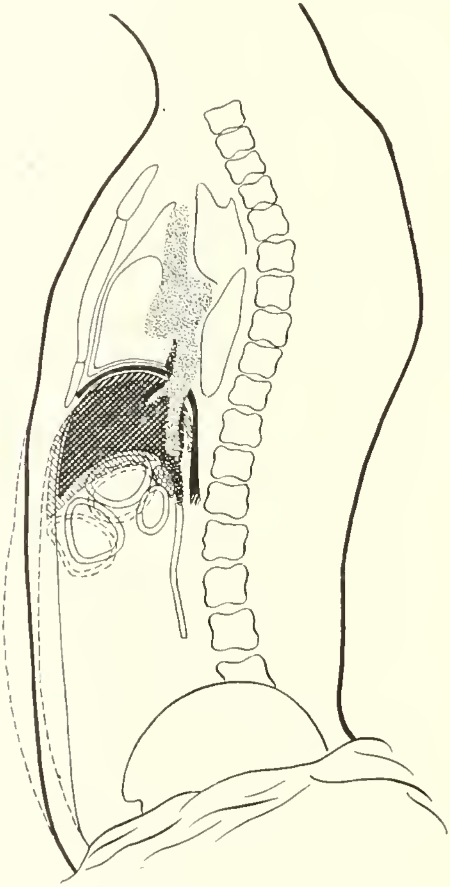

Fig. 44.—Illustrating the movements of the diaphragm and thoracic and abdominal walls as well as the change in position of the intrathoracic and intra-abdominal viscera during respiration of the abdominal type. The movements are from the solid lines on expiration to the broken lines on inspiration. (Hasse.)

44 represents the diaphragmatic type of breathing. The walls of the chest are not moved to any great degree, but the diaphragm pushes downward and the anterior and lateral walls of the abdomen are pushed outward. In its downward action the diaphragm compresses and squeezes the abdominal viscera and in this way forces the blood from the intra-abdominal organs. This effect is all the more important because the thoracic cavity is being enlarged at the same time as the compressing force is being

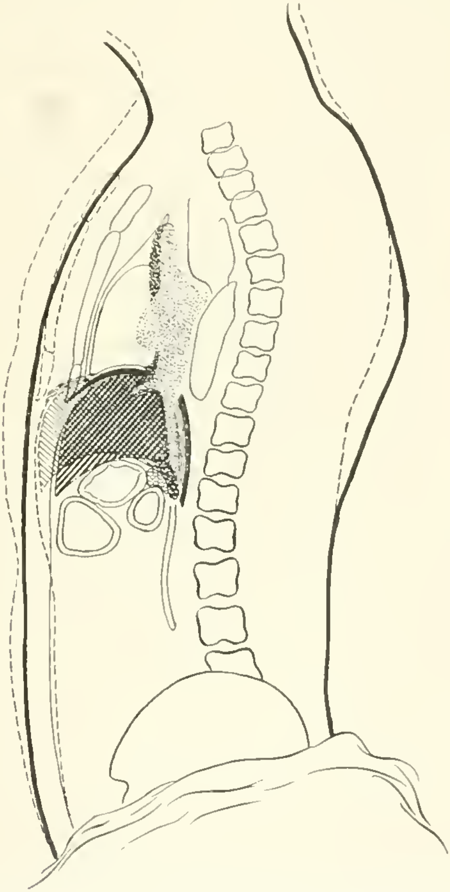

Fig. 45.—Showing the movements of the diaphragm and thoracic and abdominal walls as well as the change in position of the intrathoracic and intra-abdominal viscera when combined thoracic and abdominal breathing are pronounced. The movements are from the solid lines on expiration to the broken lines of inspiration. (Hasse.)

exerted. Fig. 45 illustrates the type of combined thoracic and diaphragmatic breathing in which the compressing abdominal force is more than in the thoracic but less than in the diaphragmatic type.

The importance of the diaphragm as a muscle of respiration may be inferred from Fig. 46, which represents a sagittal section through the body made in such a manner as to show the crus with its attachments to the lumbar vertebræ. Contraction of this portion of the diaphragm exerts a very marked force on the abdominal contents. When the diaphragm contracts the intra-abdominal pressure is increased and the abdominal muscles are

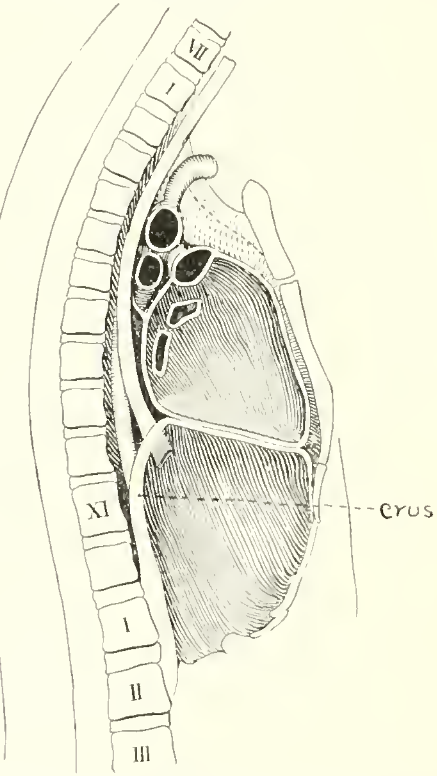

Fig. 46.—Sagittal section of the body showing the relationship of the diaphragm to the pericardium, particularly showing the importance of the crus, with its attachment to the low lumbar vertebræ, the contraction of which markedly enlarges the entire thoracic space. The crus is the portion which secures its innervation from the cervical segments of the spinal cord through the phrenics. (Wenckebach.)

pushed outward carrying the lower central arch with them, and at the same time other muscles of inspiration contract and raise the ribs. The result of this action is shown in Fig. 47.

The effect of lessening the diaphragmatic action is shown in Fig. 60, *A* and *B*, page 328. In *A* we see that as the diaphragm contracts the lower ribs are forced outward and the intrathoracic space is enlarged. In *B* we have the motion of the left half of the diaphragm lessened with a resultant decrease in the normal inspiratory enlargement of the left side of the chest. This condition is frequently met with in pulmonary tuberculosis both as a result of reflex action through the phrenics and as a result of inflammation of the pleura.

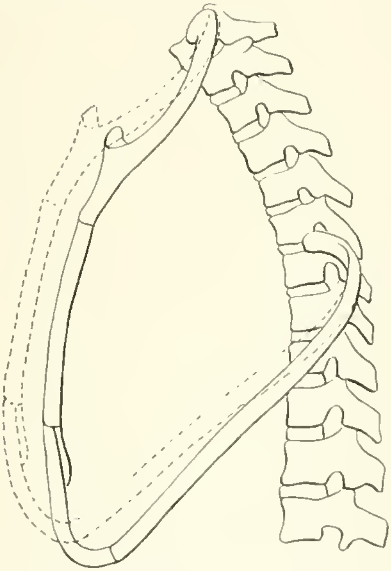

Fig. 47.—Illustrating the movement of the ribs and sternum during inspiration.

Inspiratory Act and Circulation.—One of the most important factors in the circulation of the blood, outside of the heart's action itself and the elasticity of the vessel walls, is the suction exerted upon the blood column by the thoracic cavity during respiration. The part of the diaphragm in this act can be appreciated from the following from Wenckebach:[9] "The diaphragm of mammals is the most important factor in the filling of the heart, while in amphibians and the lower vertebrates it is the only factor.''

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[9] Über pathologische Beziehungen zwischen Atmung und Krieislauf beim Menchen. Sammlung klinischer Vorträge (Volkmann) Innere Medizin, No. 140 and 141, 1907.

The pressure within the thoracic cavity outside of the air passages is negative and decreased with every inspiration. The diaphragm contracts forcing the abdominal viscera downward and the lower border of the ribs outward both anteriorly and laterally, the other muscles of inspiration contract simultaneously and lift the ribs, at the same time increasing both the anteroposterior and lateral diameters of the chest! The result in normal respiration is a very marked increase in the intrathoracic space, causing a markedly negative pressure and dilatation of the large veins and chambers of the heart. During the entire act of inspiration the blood is being sucked in to fill these veins. With every expiration, on the other hand, the negative pressure diminishes, the veins and auricles become less distended and the return flow of blood into the thorax is impeded. The intrathoracic pressure is decreased in direct proportion to the depth of the inspiration, consequently the favorable influence upon circulation likewise varies with the depth of the inspiration. Conditions which increase intrathoracic pressure embarrass the heart and may prove serious.

The following are the three chief factors concerned in the displacement of, and disturbance in the function of, the diaphragm in tuberculosis; the reflex stimulation with consequent change in tone from the inflammation in the lung, not only of the diaphragm itself through the phrenics but of the other muscles of inspiration through their respective nerves; the loss of tissue and contraction and compensatory emphysema which occurs in the lung, and the alteration of the intra-abdominal pressure due to the wasting of the intra-abdominal organs and the abdominal muscles. These factors operating either singly or jointly, as they frequently do in tuberculosis, will cause disturbance in the circulation.

Not only is an aspirating effect normally exerted upon the blood, sucking it into the thoracic cavity and heart, but, as the diaphragm contracts it increases the intra-abdominal pressure and exercises a compressing force upon the contents of the abdominal cavity and actively squeezes the blood out of the organs.

A disturbance in the action of the diaphragm and the accompanying embarrassment of the act of inspiration then has a very deleterious effect. Failing to pump the blood into the heart in its normal amount, the heart must accommodate itself to a relatively small amount of blood; also to delivering smaller amounts of blood at each systole, the result of which is:

1. A decrease in the actual size of the heart.
2. A relatively smaller amount of blood in the arteries than normal, thus producing a relative arterial anemia.
3. A decrease in blood pressure, and
4. A storing up of blood in the venous reservoirs of the body, particularly the liver, vena cava inferior and splanchnic veins.

The small heart has long been described as being a predisposing factor in tuberculosis; but it seems to me to be accounted for far more satisfactorily in this natural way.

Symptoms Following Deficient Inspiratory Act.—As a result of the storing up of the blood in the liver and splanchnic veins we have many of the digestive disturbances which are so common in tuberculosis. This congestion interferes with the function of the organs. It causes the secretions to be altered, disturbs motility, favors intestinal stasis, gas formation, and colitis; and, by retarding the blood and lymph movements, favors metastatic infection. It also interferes with the general metabolic activities throughout the body.

As a result of the various conditions which are produced by the disturbance of the function of the diaphragm, there is a certain chain of signs and symptoms which manifest themselves, most of which are of the cardioneurotic type.

These patients look pale, yet, if the blood is examined, the expected change in its constituents may not be found. This is particularly characteristic of the tuberculous patient, even early in the disease. While we must admit that anemia can and does occur in early tuberculosis, and is often an accompaniment of late tuberculosis, and that tuberculosis develops in anemic individuals; yet there is a paleness common, even in the early stages as well as the late stages, which is accompanied by little or no change in the blood picture. This is accounted for by the *relative arterial* *anemia* which results from the reflexly disturbed action of the diaphragm and the other muscles of inspiration, producing an embarrassment of the inspiratory act and a consequent lessening of the amount of blood sucked into the heart and a diminution of the amount delivered into the arteries at each systole. The systemic arteries being relatively empty, the patient appears pale.

The same condition accounts for the small pulse and low blood pressure and is another factor which tends to increase the heart's action. Shortness of breath, lack of endurance, dizziness, faintness, palpitation and coldness of the extremities are often complained of. Disturbance in function on the part of the abdominal viscera, such as altered secretions, disturbed motility, colitis, flatulence, diarrhea and constipation are very common.

One thing particularly characteristic of the abdominal complications just mentioned in tuberculosis is that they are more persistent and yield less readily to treatment. This is probably due in part, at least, in the advanced disease, to degenerations present but also to the constant congestion of the abdominal organs.

An unstable nervous system is another condition which may be traced to this cause, while there is some degree of neurasthenia in nearly all tuberculous patients, yet I have noted that there is an unusual instability often present where the diaphragmatic disturbance is marked. Especially is this noted during the time when the disturbance is acute. It is remarkable, however, to see how well nature can compensate for these disturbances. With patience and encouragement during the time when the destructive process is going on in the lung and the compensatory changes are taking place, the patient will usually arrive at a place where there is no more shifting of the position of the organs and then a remarkable degree of stability will be acquired and the troublesome symptoms will disappear.

In order to comprehend the great importance of the inspiratory act in aiding the circulation of the blood in man, and to fully grasp the part played by the diaphragm it is but necessary to study the effect of the upright position in animals. It has been shown by physiologists that small animals like the rabbit and guinea pig held in the upright position for a period of time, are unable to maintain their circulation; and die of arterial anemia. Man, on the other hand, with his well developed diaphragm and strong abdominal muscles, is able to overcome the effect of gravity and still maintain equilibrium between the venous and arterial circulation so long as the respiratory act remains undisturbed. When this is disturbed, however, he is still able to maintain the circulation but with certain difficulties in the way.

Particular Alterations in Position and Function of the Diaphragm in Pulmonary Tuberculosis.—It is probably within facts to state that the diaphragm is always disturbed in its function in pulmonary tuberculosis. This disturbance is both reflex and compensatory in character, as previously mentioned. It is rarely symmetrical, but usually more marked on one side than the other. It may be a deficiency in motion; it may be a high position on one side and a low position on the other; a high position on both sides; or a low position on both sides. When the changes are compensatory in character the position of the diaphragm depends on the relative pressures of the thoracic and abdominal cavities and on the contractions and hypertrophies which take place.

In many instances where marked displacements of the diaphragm take place on account of destructive change in pulmonary tissue, there is also a serious wasting in the tissues in the abdominal cavity together with a weakening of the abdominal muscles. This wasting tends to lower the intra-abdominal pressure and also permits the abdominal organs to assume a lower position in the abdomen (enteroptosis). At the same time it takes away from the diaphragm one of the chief forces in maintaining its normal position. Likewise the diaphragm loses its power of expressing the blood from the abdominal organs. Its action is sometimes disturbed by adhesions in the costal angle.

Resulting from such conditions we often see, instead of the normal outward motion of the lower ribs anteriorly and laterally, a drawing in with every inspiration. This is often only on one side, sometimes on both. The effect is greatly to embarrass the circulation. The aid normally due the circulation from widening the lower portion of the thoracic cavity, is taken away and a narrowing is substituted for it. At times the diaphragm is so low that the heart hangs suspended from the large vessels at the base, as mentioned above.

A high position of the diaphragm as we usually meet it, is connected with an increase of fat deposited in the mesentery, gas in the gastrointestinal tract, or some tumor mass in the abdominal cavity.

The symptoms produced by it are not as serious for the circulation as those produced by a downward displacement. In tuberculosis, however, we have other conditions present, such as the lowered intra-abdominal pressure, the weakened abdominal muscles and the pathological changes in the thoracic cavity, so the situation is different; and it must also be looked upon as being a condition extremely detrimental to a full and free respiration and as having an embarrassing influence on the circulation.

Splanchnic congestion favors acidosis by causing stagnation of the blood column.

Lessened abdominal pressure, caused by laxness of the tissues, reduced inspiratory act and weakened abdominal muscles, is followed by cerebral anemia, producing headache, dizziness, muscular weakness, and general symptoms of nervousness.

It is of great interest to see how far nature will go in her efforts at compensation. In Fig. 40, page 286, I show a wonderful compensation in which nature practically formed a new lobe to the lung and pushed it far over into the right thoracic cavity to fill the space rendered vacant by a destructive process. In the same case I mentioned how a new lobe was made to the liver which pushed far up into the thoracic cavity to occupy the space which could not be filled by the thoracic viscera. In this connection I would cite the ideas of Professor Tandler of Vienna, who speaks of the liver as a compensatory organ being able to enlarge to occupy space rendered vacant by loss of tissue.

From the foregoing description of compensatory changes which take place in pulmonary tuberculosis it is evident that the symptoms produced by them are varied in nature, and that they may be on the part of the respiratory system itself, or the circulatory, nervous or digestive system.

Effect of Arterial Hypotension and General Wasting of Tissues Upon Body Activities.—Hypotension to my mind is an important factor in the general depressed state of these patients. This has usually been thought due to the toxemia, but I believe disturbances in the diaphragm an equally important factor.[10] The disturbance on the part of the diaphragm comes early and lasts as long as activity is present if the case be an early one; and becomes permanent in all cases where destructive changes in the lung or pleural adhesions at the base result. In the case of the advanced disease, the factors, such as general wasting, degeneration of the heart in particular, and displacements of this organ, are added to the toxemia and circulatory changes caused by the disturbance of the diaphragm and coincident respiratory embarrassment.

The pulse in quiescent advanced tuberculosis where the heart is fully competent is not far from normal as long as the patient is at rest or even under light exercise, unless large amounts of the pulmonary tissue have been destroyed. If more strenuous work is engaged in, however, the heart is apt to fail to measure up, because it is already working under strain and on its reserve power. The pulse increases to a greater rate than would be normal for the work done and fails to return to the normal within the proper time limit.

Where larger amounts of lung tissue have been destroyed the patient may still be comfortable at rest; and yet not be able to do any exercise without manifesting respiratory embarrassment, the pulse being rapid, dyspnea appearing, the patient feeling weak, dizzy or faint. The reason for this is clear. The patient is ordinarily using up all of his reserve heart power and still is having difficulty in maintaining an efficient circulation. As long as he is at rest the functions of the body are carried on economically as a minimum of energy is demanded. But, when exercise is attempted and more blood is demanded the patient's heart cannot measure up to the emergency, neither can the blood be forced from the splanchnic reservoir where it is stored; consequently the symptoms follow. The flow of blood is four or five times as rapid in active muscles as in resting muscles. Rising to a position ready for walking doubles the requirements of oxygen compared with a resting position; and starting to walk quadruples it. The heart in its weakened condition and its unnatual position, with the changes in the arterial walls, and the blood stored up in the veins, cannot meet the circulatory requirements.

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[10] Enteroptosis and Altered Function of the Diaphragm, Resulting from Intrathoracic Inflammations, New York Medical Journal, December 16, 1911; and Blood Pressure in Pulmonary Tuberculosis, New York Medical Journal, August 31, 1912.

While many of these factors cannot be removed, yet there is something that can be done. The heart cannot be wholly relieved, the arteries cannot be changed, the respiratory movements cannot be restored, but the intra-abdominal pressure can be increased to a certain extent by properly applied adhesive straps or suitable abdominal binders so that the venous stasis will be somewhat relieved and the onflow of blood to the heart hastened. In this manner, circulatory embarrassment in advanced tuberculosis can be greatly relieved. An increase of fat in the mesentery in such cases is very desirable and the greatest relief is experienced by increasing the patient's nutrition to the point of putting on weight.

The following case illustrates a very marked degree of compensation and the symptoms which arise as a result of it:

Case 2414.

Housewife, age 32. Entered the sanatorium February 27, 1914.

Personal History.—During childhood suffered from diphtheria, scarlet fever, typhoid fever, smallpox, and measles.

Tuberculous History.—Five or six years prior to the time of entering the sanatorium the patient suffered from left-sided pleurisy. Two and a half years later she had a diagnosis of tuberculosis made, by the finding of bacilli in the sputum. She was sent to Denver in July, 1912, where she remained, until the time that she entered the sanatorium, with the exception of one month which she spent in Phoenix. Two years prior to the time of entering the sanatorium she was having what were considered as "colds." These were protracted and she had some hoarseness and cough. These were accompanied by sputum which showed bacilli. She also suffered at that time from night-sweats, malaise, lack of endurance, and impaired digestion. Her temperature had been irregular. At times it had been as high as 101°. Her symptoms may be grouped, according to their etiology, as follows:

THOSE DUE TO TOXEMIA.	THOSE DUE TO REFLEX CAUSE.	THOSE DUE TO TUBERCULOUS PROCESS *per se.*
Malaise	Hoarseness	Frequent and protracted colds
Lack of endurance	Cough	Pleurisy
Digestive disturbances	Pain in shoulder	Sputum—bacillus-bearing
Increased pulse rate		
Night sweats		
Fever		

Physical Examination.—Physical examination made at the time of entering the sanatorium showed a widespread disease affecting the entire left lung and the upper portion of the right. Infiltration was very denso in the upper portion of the left side and there were many râles, indicative of necrosis, throughout the entire lung. There was also evidence of cavity. There was blowing expiration, whispering voice, together with medium râles, metallic in character, near the apex posteriorly. On the right side many râles were also present, indicating that the disease in this lung had also gone on to ulceration. The left lung had contracted markedly, and the mediastinum had shifted toward the left side. The left border of the heart was in the nipple line and the right border was near the middle of the sternum. The right lower lobe was emphysematous.

Course of Disease During Treatment.—The course of the disease in this patient has been one of continuous destruction on the left side and steady improvement on the right. This patient has been under observation for thirty months, and at the present time the function of the entire left lung is destroyed. The upper lobe is replaced by cavity. There is also extensive excavation in the lower lobe, and the portion which remains is fibrous in character. The disease in the right lung has healed and the entire lung is emphysematous. Almost continually during the thirty months of observation this patient has had some rise in temperature. At no time has she been free for more than a few days. She also had fever for many months before coming under my care.

I wish to illustrate by this case one of the most remarkable compensations that I have ever observed in tuberculosis; and, at the same time, detail the symptomatology which results from the pulmonary destruction, the shifting of the mediastinum, and the shifting of the diaphragm. I shall attempt to describe this by first giving the physical examination of the patient at the present time; second, by a description of the symptoms, as they have been found throughout the course of the disease.

Present Condition on Physical Examination.— *Inspection.*—On inspection we find the patient in a high state of nutrition. Contrary to what would be expected, there is only a slight degeneration of the soft tissues recognizable. The muscles and subcutaneous tissue on the left, both anteriorly and posteriorly, appear to be somewhat wasted as compared with those on the right. The left side of the chest, as a whole, is also somewhat smaller than the right. The motion at the base is limited on the left and exaggerated on the right. A pulsation can be seen in the upper portion of the left lung, extending from the first interspace down into the axilla.

Palpation.—Palpation confirms a slight wasting of the muscles and subcutaneous tissue on the left side, as compared with the right. It also confirms the fact that the left side of the chest is somewhat smaller than the right. It shows the diminution of respiratory motion on the left and increased motion on the right. Aside from this the trapezius and levator anguli scapulæ show slight spasm.

Palpation of Total Density.—On deep palpation we find that the upper and outer portion of the left lung is separated from the chest wall by the heart, which is pushed firmly against the upper and inner axillary aspect of the bony thorax, there being almost no space between the mediastinal contents and the upper, inner aspect of the thoracic cage. The apex, or that part of the heart which produces the impulse against the chest wall, presents high in the axilla, in the fifth interspace. The right border of the heart lies fully one inch to the left of the sternum. The lower portion of the left lung is fibrous and there is no air containing tissue below the heart. The diaphragm is drawn up. Posteriorly, from the apex to the lower angle of the scapula, there is an absence of resistance, indicative of cavity. This takes in most of the portion of the lung above the lower angle of the scapula. Near the lower angle of the scapula there is a marked resistance, suggestive of dense fibrous tissue; but no air containing tissue can be found below this point.

On palpating over the right lung, increased tension, but decreased resistance is found throughout. This is also found to extend beyond the center of the thorax to a line perpendicular with the left nipple showing that the right lung pushes over to the middle of the left thoracic cavity. Palpation also shows that the lower border of the right lung has pushed downward and fills in the entire complementary space. Posteriorly, the right lung is prevented from extending beyond the median line by the posterior mediastinum; but its inferior margin extends to the 12th rib.

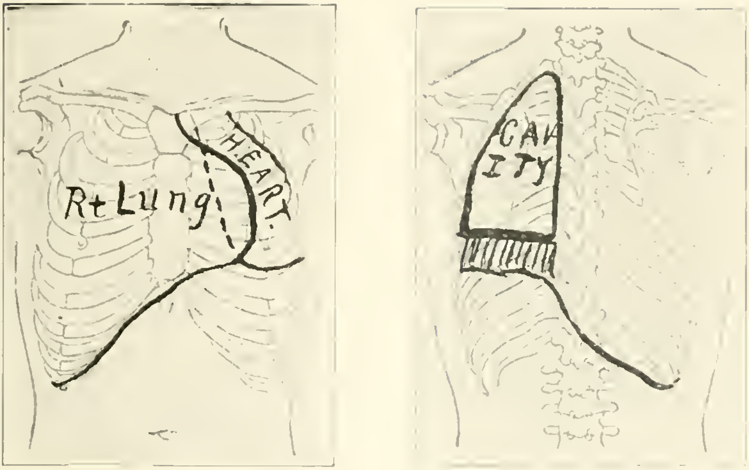
Percussion and *Auscultation* confirm these findings.

The condition, as observed, is schematically represented in the accompanying Fig. 48, *A* and *B*.

Symptoms.—Aside from the usual symptoms which accompany chronic active tuberculosis, this patient has suffered from those which belong to the cardioneurotic group; thus, tiring, lack of endurance, exhaustion on the least effort, dizziness on assuming the erect position, dyspnea on exertion, insomnia, and variable appetite. These symptoms are indicative of an inability of the patient to furnish an adequate amount of blood for the body activities. The blood pressure in this patient is very low, being in the nineties, and has been found below ninety. She has a very pronounced splanchnic congestion; her heart, while not markedly rapid when at rest, is quite rapid on exertion. As long as this patient retains the prone position her functions are carried on fairly well; but, as soon as there is an extra demand made upon her, as by exertion, she is unable to supply the extra blood that is required; consequently, she suffers from the symptoms mentioned above. When she assumes the erect position she has a cerebral anemia, which causes dizziness and a feeling of faintness.

It will be seen, from the examination findings above, that this patient's diaphragm is working at a disadvantage. On the left side it has assumed a very high position, and on the right side a very low position, consequently the force of the inspiratory act is very much diminished. Resulting from this diminution of the inspiratory act, the venous reservoirs throughout the body are in a state of engorgement, while the arterial system is deficient in blood.

Another factor which operates to increase the relative splanchnic congestion is the lowered intra-abdominal pressure, due to enlargement of the cavity by the high position of the diaphragm on the left side. While this is compensated to a great extent, by the low position on the right, it is not fully compensated; consequently, there is a deficiency in substance within the abdominal area, which results in lessened pressure, and,



A *B*

Fig. 48.—Showing schematically the compensation which has taken place between the two sides of the chest, and between the thoracic and abdominal cavities. *A*, anterior view; *B*, posterior view.

consequently, lessened tone of the vessels. Normal exercise calls for an extra amount of blood, and this is supplied from the venous reservoirs, particularly those in the splanchnic area. Now, owing to the deficient inspiratory power possessed by this patient, she is unable to aspirate the blood from the veins into the right heart and furnish the required blood, consequently she suffers from a lack of endurance, an inability to perform physical work and dyspnea.

Treatment.—The respiratory equilibrium of this patient cannot be restored. The functional capacity of the diaphragm will always be limited. The splanchnic congestion is more or less permanent; yet, there are certain things which can be done which will partially restore the circulatory equilibrium in this patient, and give her a fair degree of endurance. This must come about through strengthening of the muscles of the body, depositing a certain amount of fat in the abdominal cavity, and increasing intra-abdominal tension. Exercise would aid this patient by strengthening the body muscles and affording firmer support for the vessels. This is difficult of attainment in this case because there is often a slight rise of temperature present, so that, as soon as we make a certain amount of headway through exercise, she is compelled to assume the prone position again.

Such patients must be kept in good nutrition for the intra-abdominal fat is desirable; yet this must not be carried too far for it will increase dyspnea and flabbiness.

Another feature of value in cases such as this is an abdominal support, which will increase intra-abdominal pressure. These cases are not easy to handle, and yet a great deal can be done for them. If the activity of the disease in this case ceases, I think it is probable that a fair degree of endurance may be attained. As soon as better tone can be obtained in the body, and particularly the abdominal muscles, this, if accompanied by increased intra-abdominal fat, and reenforced by an abdominal support, will aid in pressing the blood from the splanchnic vessels.

Two years later, this patient is free from fever, has regained her normal weight, and is able to walk two miles a day, going slowly, without tiring.

CHAPTER XII.

TRAUMATIC TUBERCULOSIS.

The relationship of trauma to tuberculosis deserves attention. The medico-legal aspect of this question at times assumes importance. Many cases of tuberculosis have been reported in literature in which trauma is supposed to have been the primary cause of the disease. In former times, when we did not understand the nature of tuberculosis as well as we do today, it was more difficult to determine the definite relationship between a previously received trauma and an active tuberculosis. Today, however, knowing the nature of tuberculosis as we do, knowing that bacilli circulate in the blood more or less frequently after infection has once occurred; and realizing further that implantation of bacilli may be favored by a traumatic condition; and, further, realizing that an old quiescent focus might be disturbed in such a way as to mobilize bacilli through trauma, we can see that several different conditions might arise whereby there would be a direct relationship between the trauma and tuberculosis. We cannot conceive of trauma itself, unless it be a puncture by a bacillus-infected instrument, producing tuberculosis. Infection must either be present prior to the time of the trauma, or conditions must be produced as a result of the trauma which favor some future implantation.

To further understand the relationship of trauma to active tuberculosis, it is necessary to bear in mind that a very large percentage of adults have quiescent foci of tuberculosis within the thoracic cavity. They may be in the glands or they may be in the lung tissue itself. The postmortem reports of Hart, referred to elsewhere in these pages, show that more than fifty per cent of adults have tuberculous infections in the pulmonary tissue; but by far the greatest proportion of these will remain quiescent during the life of the patient. However, if disturbed by trauma, a quiescent focus may be caused to take upon itself activity, and produce clinical tuberculosis. The effect of trauma in mobilizing bacilli may be readily appreciated by recalling how such organs as the liver and spleen are often ruptured as a result of a blow over them.

As physicians, it is our duty to understand this question in order that we may be able to render a fair judgment in cases which come to us for an opinion.

From what has previously been said it can readily be understood that a variety of conditions might arise whereby it would be necessary to determine the relationship between trauma and existing active tuberculosis. The problem is rendered more difficult because many patients suffer from active tuberculosis for a prolonged period without having a diagnosis made.

Trauma might be either the actual cause of liberating bacilli from a previously latent focus or of causing a spread and increase in severity of active disease which is already present; or, it may injure the tissues and favor infection in a part of the lung tissue which had not previously been infected. In each of these instances the trauma will stand in a causative relationship. In the first, it must be looked upon as the precipitating cause of clinical tuberculosis. In the other instances it would be looked upon as having an influence in increasing the severity of already existing clinical tuberculosis, and not as standing in the relationship of producing clinical disease. Therefore, before determining the relationship of trauma to an existing active tuberculosis, it is necessary to know the antecedent history of the patient. One should know whether an active tuberculosis had previously existed. In forming an opinion as to whether or not a previous clinical tuberculosis had existed, we would be obliged to base it upon the presence or absence of clinical history of some previous attack of tuberculosis, or a clinical history of suspicious symptoms immediately prior to the time of injury.

A patient who had previously suffered from a more or less active tuberculosis, but who had attained a healing of the same, should be looked upon in the same light as a patient who was previously suffering from a quiescent unrecognized lesion. Such an individual would be more apt to be seriously injured by trauma than an individual who was suffering from a small lesion. If the patient who had previously suffered from clinical tuberculosis, was well and had been free from symptoms for months prior to the injury, we would then be compelled to look upon the trauma as standing in direct causative relationship to an active lesion following injury. In this connection, however, we must bear in mind that lesions that are extensive, even though healing has been attained, now and then show evidence of becoming active again in after years; but, if activity had not manifested itself in any clinical symptoms or physical signs prior to the trauma, we would consider the trauma as being the active factor in the production of the clinical disease.

For cases of active clinical tuberculosis to be definitely assigned to trauma as the agency in their active causation, the symptoms must appear promptly following the injury. The spreading must take place within the time that implantation and the development of tuberculosis would usually occur,—that is within a period of a few weeks. In this connection we must realize that it is possible for bacilli to be mobilized in small numbers and produce very small metastatic foci. Clinical tuberculosis might result from such an infection, yet require a considerable time for the bacilli to be implanted and form new metastases; and, in such cases, it might be difficult to give an accurate opinion. In cases, however, where the symptoms show themselves within a few weeks after the injury, the relationship can hardly be doubted. That a previous focus may, as a result of trauma, assume activity, which results in the escape and scattering of bacilli to new tissues, is entirely reasonable. On this point so able an authority as J. Orth[1] makes the important statement that crushing or concussion of the thorax might produce such a marked injury to the tissues, even those which were not lying immediately under the point of trauma, that mobilization of bacilli in a quiescent focus might take place.

The truth of this fact is impressed upon us in the experiments which are being reported from soldiers in the present war. It is not uncommon to find tuberculosis follow immediately a gunshot wound of the thorax. This can be explained on the ground that a projectile, in passing through the pulmonary tissue, produces a trauma in tissues which have previously been infected, and thus causes an escape of the enclosed bacilli with resultant formation of new foci. This can occur either in quiescent lesions or lesions that are active. There is quite a large percentage of gunshot wounds of the thorax which are followed by active clinical tuberculosis.

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[1] Trauma und Lungentuberkulose Vier Obergutachten erstattet von Geheimrat, Zeitschrift für Tuberkulose und Heilstätenwesen, Bd. xxv, Heft. 1, 1915.

I have seen several cases of supposed traumatic tuberculosis, but, in most instances the injury had been received so long prior to the time of the active disease that no connection could be established. In most instances, the disease was at the apex while the trauma had been elsewhere. The diagnosis of traumatic tuberculosis had been made simply because the patient had previously received an injury to the chest.

The following case illustrates how injury to the chest, producing contusion of tissues may increase the symptoms and produce a more rapid course in a patient who was previously suffering from active tuberculosis. Further, it will be noted, that it is quite probable that the injury to the tissues in this instance produced a condition which favored implantation in a new area.

The patient was a man of about thirty years of age. He received an injury by stumbling and falling on a piece of iron, which caused a fracture of the sixth rib on the left side in the anterior axillary line. The rib punctured the lung, and was followed by spitting of blood. Traumatic pleurisy followed at once.

I examined the patient within two months after the occurrence of the hemorrhage, and found a localized tuberculous process surrounding the point of injury. The inflammatory process was the seat of marked activity, causing toxemia with rise of temperature. This patient had previously been suffering from a tuberculous process of the left apex, moderate in extent, and slightly active.

The weak point in the argument in this case is that I had not examined the patient prior to the time of his injury, and cannot personally vouch for the fact that there was not an old lesion at the point of injury. However, he had been examined shortly before receiving the injury by a competent man, who found no evidence of trouble at the base. The fact that there was an area entirely free from involvement between the lesion at the apex and the more extensive one at the base, together with the fact that the active disease at the site of the trauma, apparently came on promptly after the injury, led me to believe that this was probably a new localization in which implantation of bacilli had been favored by the contusion of the tissues. I am not unmindful of the fact, however, that there might have been a small quiescent focus in the tissues at the point of injury, from which bacilli escaped as a result of the contusion.

In this case, we cannot look upon trauma as being a direct cause of the clinical tuberculosis, but we are forced to the conclusion that it was, in all probabilities, the cause of the new focus at the base of the lung, and its relationship to the disease in question was one of increasing its activity and hastening its progress.

The next case answers all the requirements necessary for a diagnosis of traumatic tuberculosis. Evidence is present that there was an old lesion affecting the hilus and the apex of the left lung. From this, bacilli were mobilized by the trauma received at the time of the accident. Prior to the time of the injury, the patient was in perfect health, and gave no history of ever having shown symptoms indicative of active tuberculosis. Immediately following the injury, the symptoms of active tuberculosis appeared and the diagnosis was confirmed by the presence of physical signs in the chest, the x-ray, and the finding of bacilli in the sputum.

Case 2966.

Clinical History.—Female. Always strong and robust. Lobar pneumonia running typical course when fifteen years of age. Mumps during college life. Operation on turbinates for occlusion of the nares two years ago. Otherwise no illness until, following an automobile accident which occurred on Oct. 20, 1915, the patient was rendered unconscious and received severe injuries to the lower right chest and over the left shoulder. A slight hacking cough followed immediately, apparently due to the trauma to the pleura. This persisted. Shortly thereafter, about November 13, an x-ray plate showed fluid in the right pleural cavity. The patient's cough continued and increased. The sputum which first appeared December 1, examined December 6, 1915, showed tubercle bacilli. Repeated physical examinations showed distinct tuberculous involvement.

Physical Examination.—Examination made about six months after the accident showed the following:

The patient is well nourished.

Inspection reveals somewhat diminished motion at both bases, more marked anteriorly on the right side, owing to pleural inflammation; but posteriorly more marked on the left.

Condition of the muscles and subcutaneous tissue.—There is a slight degeneration of the subcutaneous tissue near the hilus on the right side; also slight degeneration of the sternocleidomastoideus, and upper portion of the pectoralis. The right sternocleidomastoideus also shows marked tone. There is some question whether it could be in reflex spasm, or be a part of the generally well developed muscular condition resulting from continuous use. The left sternocleidomastoideus, scaleni, and upper fibers of the pectoralis show marked spasm. Posteriorly, the left trapezius, and levator anguli scapulæ are slightly degenerated, indicative of an old lesion. The same muscles, with the rhomboidei, are in marked spasm, indicative of new activity. Apparently no spasm on the right side posteriorly.

Deep palpation reveals an increased density to the 4th rib anteriorly on the left and in the region of the hilus on the right. Posteriorly, there is an increased density to the middle of the scapula on the left and in the interscapular region on the right.

Percussion on the left shows slight dullness to the 4th rib anteriorly near the sternum and to the 3rd rib toward the axillary line. There is slight impairment near the sternum on the right; posteriorly, impairment with slight dullness to the middle of the scapula on the left, and in the interscapular region on the right.

Auscultation reveals a roughened note accompanied by numerous medium and fine râles from the apex to the 4th interspace on the left, more marked near the sternum than near the axillary line. No râles on the right side, but slightly altered breathing near the sternum. Posteriorly, there is roughened breathing, with medium and fine râles extending to the middle of the scapula on the left side, with altered breathing near the vertebræ on the right in the interscapular space. There are a few fine râles which seem to be of pleural origin in the lower right anteriorly.

Diagnosis.—Pleurisy of traumatic origin at right base. Pulmonary tuberculosis. Thickening about the hilus of the lung on the right, with slight extension toward the apex. Old foci in the upper portion of the left lung, upon which an active process has been engrafted, most probably as a result of trauma. Active tuberculosis in the upper half of the lung.

Comment.—The physical condition of the patient prior to the accident; the absence of clinical symptoms; and the fact that she was able to carry on the difficult and strenuous work incidental to her professional life, without even being tired, or showing any other symptoms that might be attributed to an active tuberculous lesion, indicates that any infection that was present prior to this accident was quiescent. The indications, however, are that there was an old hilus infection present, and also a quiescent focus in the apex of the left lung. This is indicated not only by the character of the breathing on auscultation, but by the degeneration of the muscles and soft tissues covering this apex. This condition, however, is not inconsistent with good health, as is shown by such pathological reports as those of Hart, which show that in all autopsies made on adults dying of ordinary causes, an apical tuberculosis is present in more than fifty per cent. The great majority of these infections are quiescent and produce no recognizable symptoms during the life of the patient. As long as the patient remains in good physical condition, he seems to be free from the danger of the infection taking upon itself activity.

The clinical course of the infection, following the accident, is extremely interesting. Immediately following, there was a traumatic pleurisy at the right base. There was also a dry cough which, at first, was thought to be referable to the pleurisy; but the pleurisy soon disappeared, while the cough continued to increase in severity. The examination of the chest about the middle of November revealed no physical signs of active tuberculosis. Such could not be expected at this early date because an infection resulting from an activity lighted up by such an accident would most likely require a longer period to produce recognizable symptoms and signs. By the first of December the cough had still increased in severity and expectoration appeared which, on the 6th day of December, was shown to contain tubercle bacilli. Physical examination at this time also revealed an active tuberculous lesion in the upper left lung. The whole clinical course in this case seemed to point definitely to the accident as being the cause of the activity.

CHAPTER XIII.

IMPORTANT ANATOMICAL AND PHYSIOLOGICAL FACTS TO BE CONSIDERED IN MAKING PHYSICAL EXAMINATION OF THE ORGANS WITHIN THE THORAX.

In the course of time the literature of any subject becomes a mixture of truth and error in spite of the closest scrutiny. To this rule, the diagnosis of diseases affecting the organs within the thoracic cavity is no exception.

Aside from the logical analysis of signs and symptoms, if we would make more accurate diagnoses it is necessary not only to improve our technic of making examinations, but also to gain a more accurate conception of the parts and organs to be examined. This statement holds for anatomical and physiological as well as pathological considerations. A familiarity with the following points will show some of the common sources of error in physical examination.

Projection of Lung on Anterior Surface of Chest.—From the midsternum to the acromial end of the clavicle above and to the external border of the axillary fold lower down, is a distance of some six inches in ordinary chests. One must not think, however, that the underlying lung extends from the midsternum to the acromion or the axillary fold. The external border of the lung is bounded by the inner surface of the ribs. The inner surface of the first rib does not extend beyond the inner fourth of the clavicle; the inner surface of the second, not beyond the inner third of the clavicle; and the inner surface of the third, not beyond the inner half of the clavicle, consequently, the anterior surface of the lung is small in comparison with the entire anterior surface of the chest wall as may be seen in Figs. 49 and 50. The latter being a frontal section gives a splendid idea of the relative amount of lung which presents. One should bear this in mind in making examinations. The ear or stethoscope is near lung tissue only over the inner third of the interspace immediately below the clavicle and the inner half of that next lower. Above the clavicle, the apex is found between the two heads of the sternocleidomastoideus, and does not extend out in the supraclavicular notch as is usually taken for granted.

Normal Border of Lungs.—The normal borders of the lungs are shown in Figs. 51, 52, and 53. The apex of the lung extends 3 to 5 cm. above the clavicle. The lower borders are at about the same height on the two sides. For the man in middle life they are on a level with the sixth rib in the mammary line, the eighth in the axillary line, the tenth in the scapular line, and at the junction of the eleventh rib with the vertebra at the vertebral border. (See pages 322, 323, and 324.)

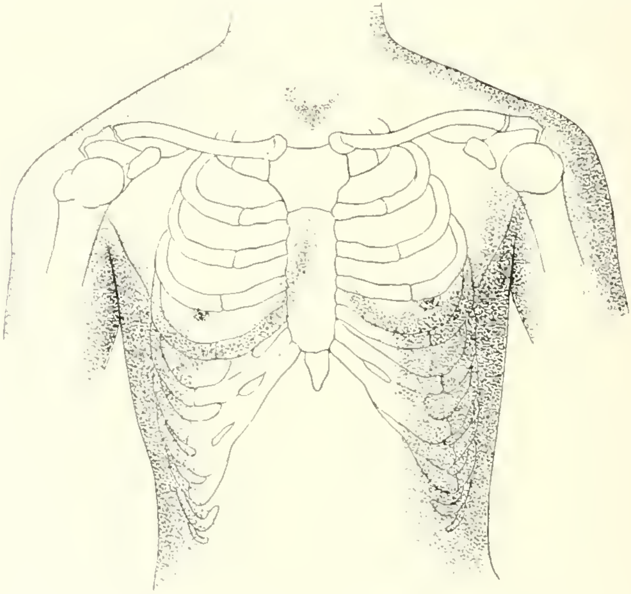

Fig. 49.—Showing the relationship of the anterior surface of the lung as confined by the bony thorax to the soft structures forming the anterior surface of the chest.

The apex is the nearest the surface between the two heads of the sternocleidomastoideus, as before mentioned. On ordinary quiet respiration the movement of the lower border of the lung is scarcely to be determined by percussion and palpation, being only 1 to 2 cm.; but the difference between forced inspira-

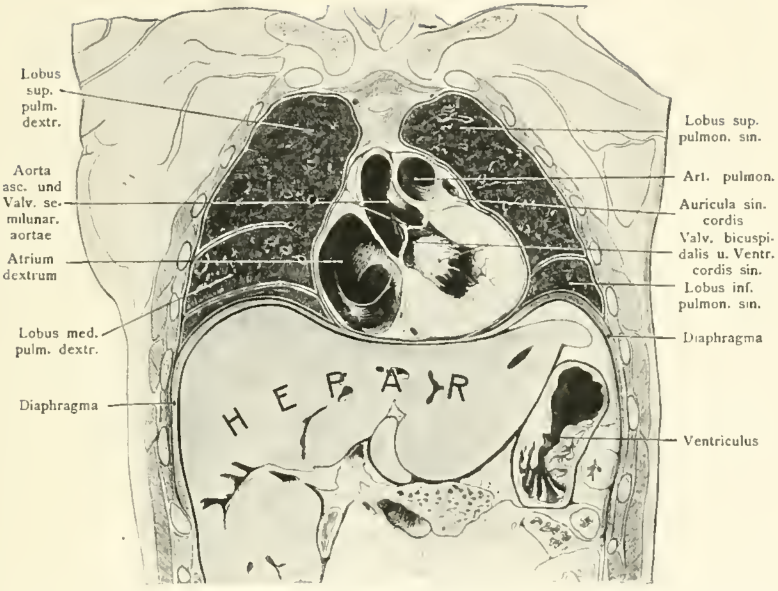

Fig. 50.—Frontal section through thorax of 26-year old man. Section made midway between mammary and axillary line. (Viewed from the front.) The thickness of the soft tissues covering the lung may be seen and their influence on palpation, percussion, and auscultation may be inferred from this figure. It also shows the relationship of the lungs themselves to the anterior surface of the chest wall. (Corning.)

tion and forced expiration can be readily determined for it amounts from 6 to 10 cm.

Position of Diaphragm at Different Age Periods.—The position of the diaphragm varies with the age of the patient. Figs. 54, 55, and 56, page 324 (Mehnert), show the diaphragm at birth, at 36 years of age, and at 72 years of age. In development, the diaphragm comes from high up in the cervical region and consequently receives a portion of its innervation from the cervical portion of the cord; the central portion being supplied by the phrenics which take their origin from the third and fourth or fourth and fifth cervical segments. This fact is important in explaining the pain which is found in the shoulder areas supplied by nerves from the third and fourth cervical segments, in cases of diaphragmatic pleurisy, when the central portion of the organ is involved. The costal portion of the diaphragm, on the other hand, is supplied by the lower six intercostal nerves, which also supply the muscles and skin of the upper abdominal wall. This fact accounts for the rigidity and pain over these parts in diaphragmatic pleurisy when the costal portion is inflamed.

Position of Sulci Which Separate Lobes.—The importance of understanding the position of the sulci which divide the lung into lobes is not so great in early cases as it is in more extensive ones. Tuberculosis extends in the lung most readily by forming metastases in lymph spaces adjacent to the original focus; or by metastatic infection through the bronchi. Extension through lymphatic metastases is confined, for the most part, to the areas adjacent to the previous focus, and does not readily leap from one lobe to the other. Bronchogenic infection, for the most part, follows the same course, but it may, like hematogenic infection also go from one lobe to the other, or from one lung to the other.

The divisions between the lobes is shown in Figs. 51, 52, and 53. Clinically, the division between the upper and lower lobe may be roughly marked by placing the hand upon the opposite shoulder, which throws the lower point of the scapula out toward the axilla, and drawing a line along its inner border. This line when prolonged, as in Fig. 57, page 324, roughly marks the division between the lobes.

In advanced tuberculosis the greatest destruction of pulmonary tissue usually occurs in the upper lobes, and is followed by contraction. A compensatory emphysema follows in the lower lobe and in the other lung, causing their enlargement. In consequence of these changes there is a shifting of the trachea, interlobular septum, mediastinum, and diaphragm. It is not infrequent in advanced cases with marked destructive processes in one upper lobe, to find it contracted to a small fibrous mass and the lower lobe anteriorly pushing up toward the apex. Such a case is shown in Fig. 40, page 286, where the middle lobe is shown as a fibrous string and the lower lobe pushed upward to the third rib. In this case the compensation was so great that

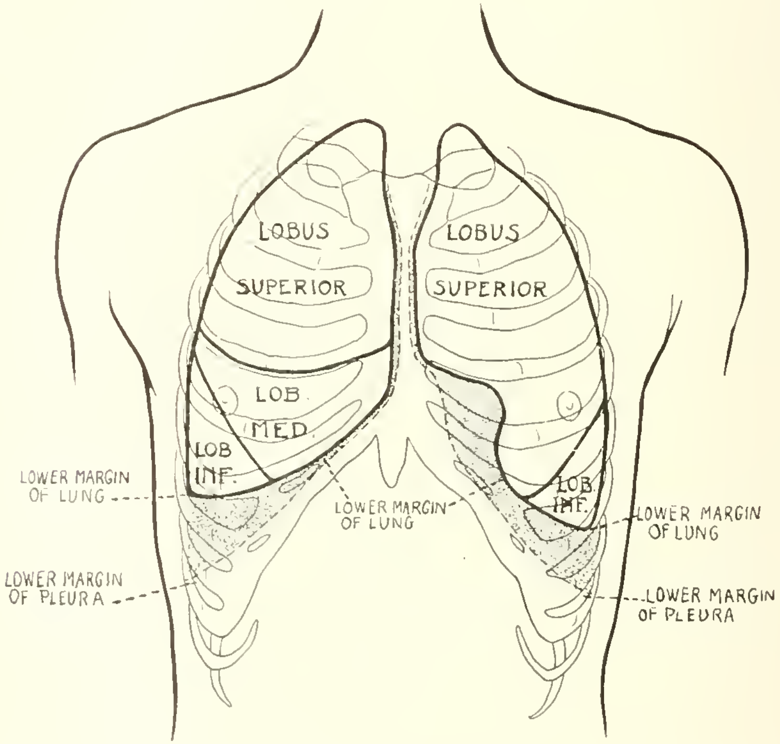

Fig. 51.—Illustrating the normal borders of the lungs and the location of the interlobular septi. Anterior view. (Corning.)

most of the right side of the chest on the anterior surface as well as the left was occupied by the left lung.

The Projection of the Peritracheal and Peribronchial Glands on Body Surface.—The projection of the peritracheal and peribronchial glands on the surface becomes of great importance now that hilus infection has assumed such great proportions. Fig. 15, page 94, shows the location of the tracheal and bronchial lymph glands which are so commonly infected and enlarged. Figs. 58 and 59 show that dullness due to enlargement of such glands in adults would be usually expected in the interscapular spaces from the third to sixth thoracic vertebræ. Dullness from

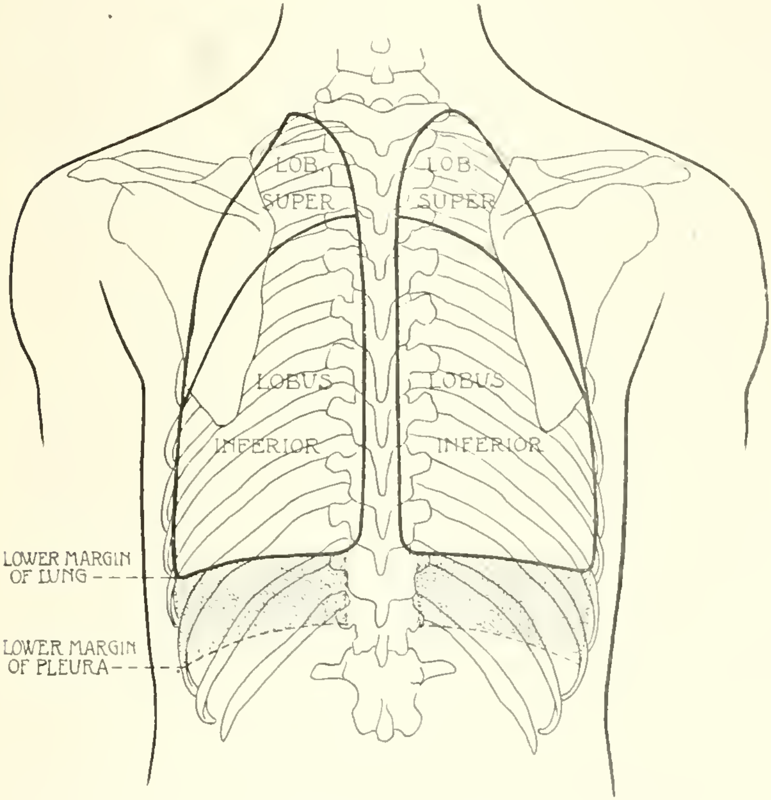

Fig. 52.—Illustrating normal borders of the lungs and interlobular septi. Posterior view. (Corning.)

these glands is most common in children, however, and is found higher than in adults. Mehnert[1] shows the changed position of the bifurcation of the trachea according to age. At birth it is at the third or between the third and fourth, or at the fourth thoracic vertebra; at two years of age it is between the fourth and fifth; at thirty-five between the fifth and sixth; at sixty in the middle of the sixth; and at seventy-two on a level with the upper third of the seventh. The mediastinal glands are nearer the posterior surface than the anterior, consequently are more readily detected posteriorly. Anteriorly, the bifurcation of the trachea is usually given as taking place on a level with the junction of the second costal cartilage with the sternum, but from data quoted above its position must be variable. (See pages 326 and 327.)

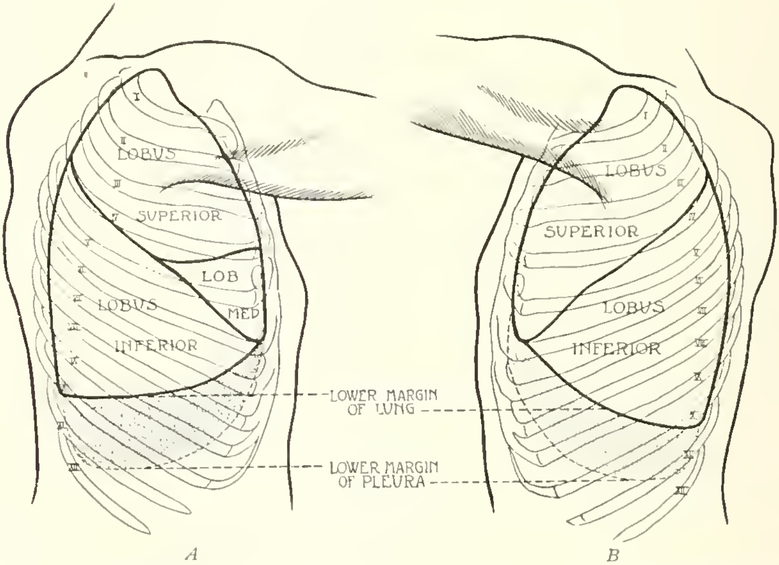

Fig. 53.—Illustrating the normal borders of the lungs and the location of the interlobular septi. Lateral view. *A*, right; *B*, left. (Corning.)

Muscles Employed in Normal Respiration.—Inasmuch as deficient motion of the chest wall is a very important and almost constant accompaniment of inflammation in the lungs and pleura; and, inasmuch as some disturbance on the part of the motion of the chest wall is usually found in both active and healed lesions

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[1] Über topographische Altersveränderungen des Atmungsapparates, Gustav Fischer, Jena, 1901.

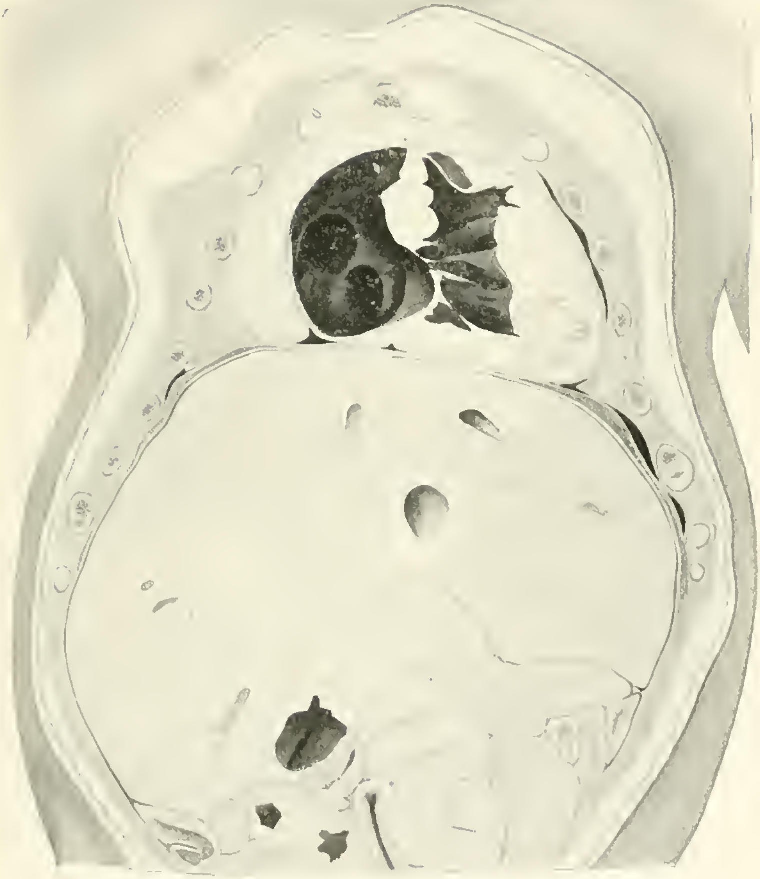

Fig. 54.—Position of the diaphragm and intrathoracic and abdominal organs at birth. Compare with Figs. 55 and 56. (Mehnert.)

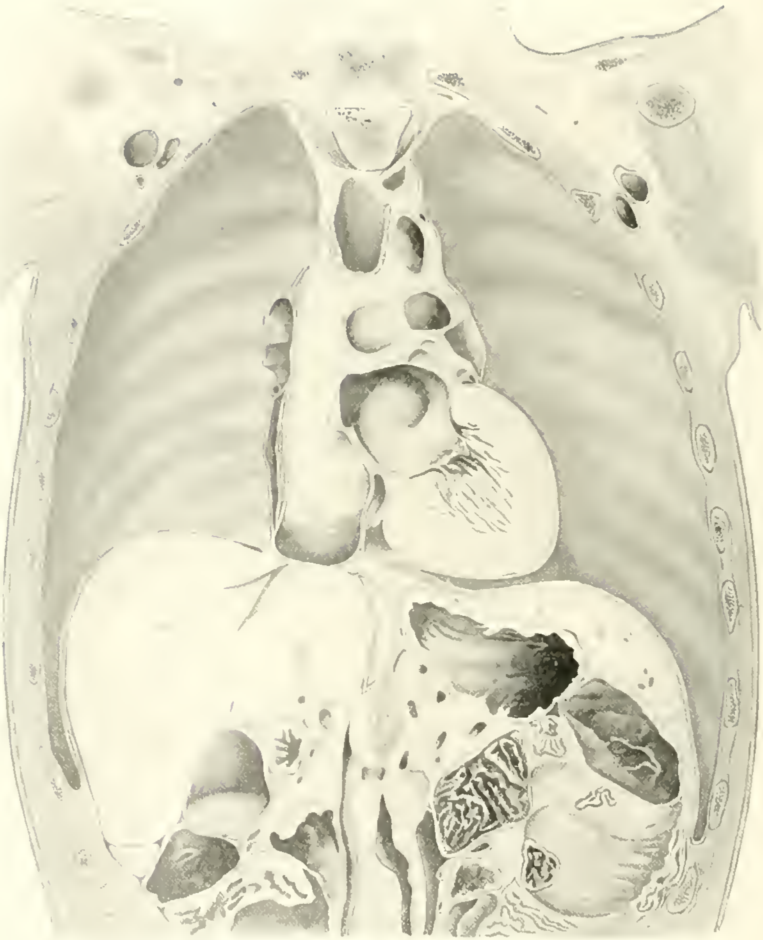

Fig. 55.—Position of the diaphragm and intrathoracic and abdominal organs in adult 36 years of age. Compare with Figs. 54 and 56. (Mehnert.)

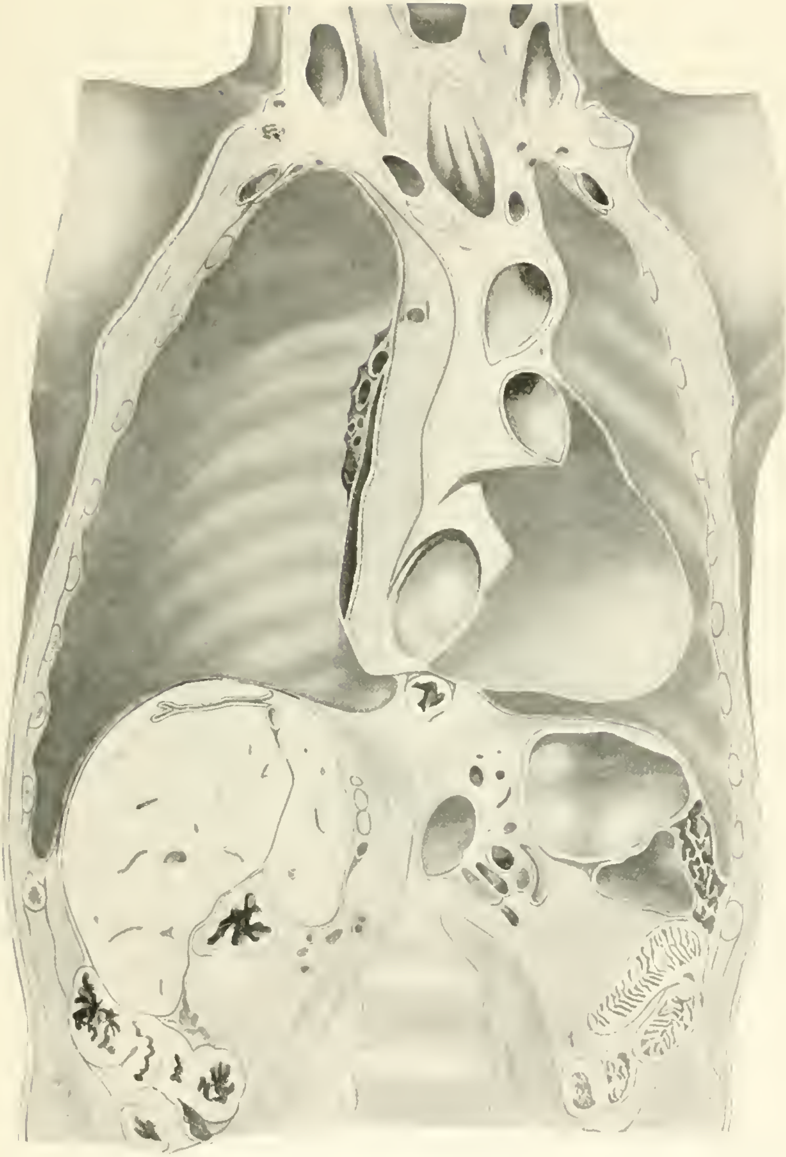

Fig. 56.—Position of the diaphragm and the intrathoracic and abdominal organs in adult 72 years of age. Compare with Figs. 54 and 55. (Mehnert.)

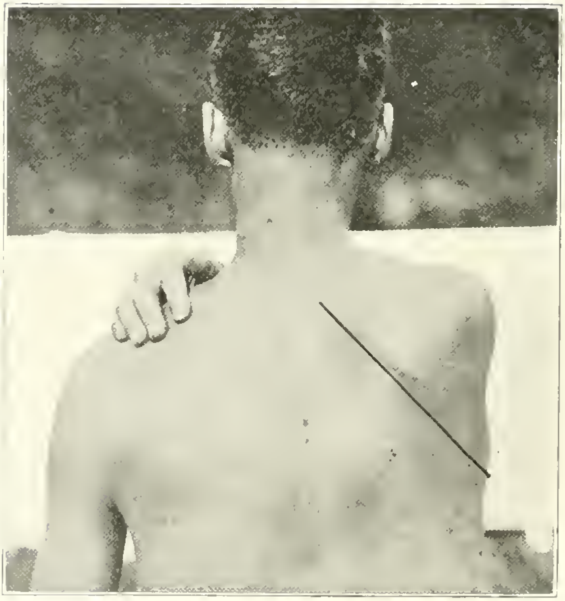

Fig. 57.—Showing the location of the sulcus between upper and lower lobes as determined by placing the hand on the opposite shoulder and prolonging a line drawn along the inner border of the scapula.

of the lung and pleura, it becomes important for the examiner to acquaint himself carefully with the mechanics of respiration and familiarize himself with the normal movements.

The inspiratory act prepares the passages (nose and larynx) for the entrance of air and enlarges the thorax anteroposteriorly, laterally and superoinferiorly. Normal inspiration is produced by the contraction of the following muscles:

1. Muscles of nose (not in quiet respiration as a rule in man) innervated by nervus facialis (seventh).
2. Muscles of the larynx—innervated by nervi laryngei superior et inferior (tenth).
3. The diaphragm innervated by nervi phrenici from the third and fourth or fourth and fifth cervical segments; and the seventh to twelfth intercostals.
4. Intercostales externi et intercartilageni innervated by all thoracic segments of the cord.
5. Levatores costarum longi et breves innervated by filaments from the spinal nerves.

The sinking of the diaphragm is the more important factor in the male while the elevation of the ribs is more important in the female. On forced inspiration, however, all muscles are brought into play and the chest is enlarged in all its dimensions.

Influence of Diaphragm on Respiration.—When sleeping or reclining, if lying on the back, both men and women assume the thoracic type of respiration. When lying on the side, on the other hand, they assume the abdominal type. The diaphragm in its innervation is reflexly associated with the sympathetics which supply the lung. The diaphragm, in common with the muscles of the shoulder girdle, is innervated partly by cervical nerves (the phrenics). The phrenics receive impulses from the inflamed lung over the upper five or six thoracic sympathetics, and intraspinal neurons which receive and transfer them upward in the cord. These reflex-relationships cause a motor reflex to be expressed in the diaphragm when the lung is inflamed and a sensory reflex in the cervical nerves, particularly the third and fourth, when the pleura covering the diaphragm is inflamed. The reflex disturbance in the movement of the diaphragm through the phrenics causes a limited motion on inspiration which becomes a valuable diagnostic sign in inflammations of the lung. It also becomes important because of its influence in lessening the inspiratory act. This, as shown elsewhere (page 301), lessens the suction action which delivers the blood to the heart, consequently, delivers less blood to that organ which in turn accommodates itself to a lessened quantity and becomes smaller. From a lessened output the arteries also contain less, showing an arterial anemia, and giving a lower blood pressure, while the veins are full, giving a venous congestion. This latter does not particularly manifest itself in the early part of the disease while methods for compensation are still available; but, later, it shows throughout the body. Congestion of the abdominal vessels is especially pronounced, for the suction action of the normal inspiratory act is lessened, and they are also deprived of the full compressing force of the contracting diaphragm upon the abdominal viscera. Wenckebach has aptly compared the compres-

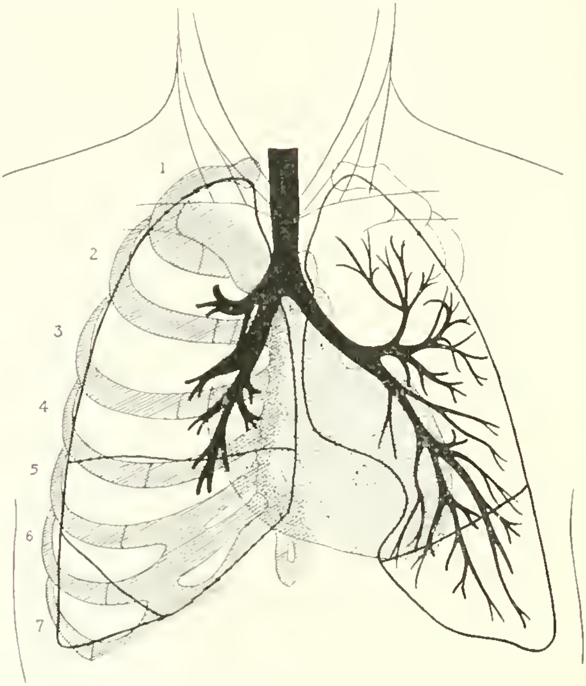

Fig. 58.—Showing the position of the bifurcation of the trachea and the peritracheal and peribronchial glands projected upon the anterior surface of the chest in a young adult. (Gerhartz.)

sing action of the contracting diaphragm upon the liver to that of the compression of a hand on a sponge.

Anything which interferes with the inspiratory act produces the effect above described. This may be understood by Fig. 60*A* and *B*. When conditions have arisen in the chest which make it necessary to call the accessory muscles of respiration into use, any influence interfering with their action will also affect the force of the inspiratory act. (See page 328.)

Muscles Employed in Forced Respiration.—Spasm of the accessory muscles of respiration becomes an important factor in the diagnosis of tuberculosis because of the fact that they take their nerve supply from cervical nerves which mediate with intraspinal neurons which receive and transmit upward to them impulses which come from the inflamed lung, the same as just mentioned for the diaphragm.

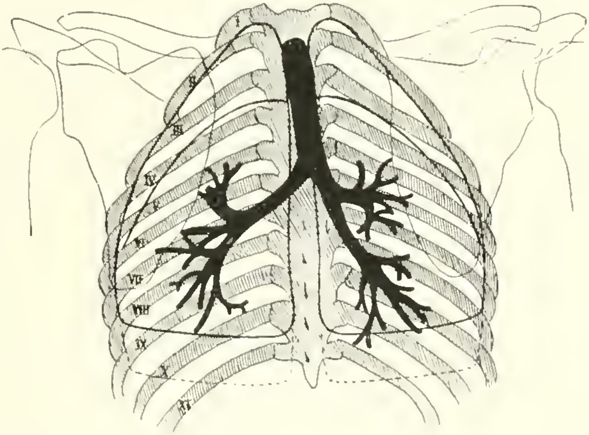

Fig. 59.—Showing the position of the bifurcation of the trachea with the peritracheal and peribronchial glands projected upon the posterior surface of the chest in a young adult. (Piersol.)

This fact will be evident from the following table of accessory muscles of respiration and their innervation:

1. Scalenus anticus, medius and posticus,—innervated by filaments from the cervical and brachial plexuses.

2. Serratus posticus superior, innervated by nervus dorsalis scapulæ from the fifth cervical nerve.

3. Sternocleidomastoideus innervated by ramus externus

nervi accessorii and filaments from the second and third cervical nerves.

4. Trapezius innervated by ramus externus nervi accessorii and third and fourth cervical nerves.

5. Rhomboidei innervated by dorsalis scapulæ from the fifth cervical nerve.

6. Extensores columnæ vertebralis innervated by posterior branches of the spinal nerves.

7. Pectoralis major, innervated by external and internal anterior thoracic nerves from the fifth, sixth, seventh and eighth cervical and first thoracic nerves.

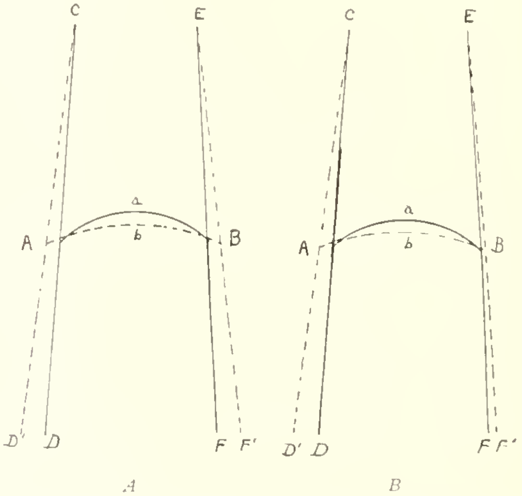

Fig. 60.—Schematic illustration of the influence of the diaphragm in enlarging the intrathoracic space. *A*, normal respiration; *B*, illustrating the effect when the movement of one side of the diaphragm is lessened. The intrathoracic space fails to be enlarged to the extent that motion of the chest and abdominal wall *EF*, in figure *B*, is limited.

8. Pectoralis minor, innervated by seventh and eighth cervical and first thoracic nerves.

Thus it is clear that these muscles, together with the diaphragm, are innervated by fibers from the cervical segments of the cord which are in communication with and receive afferent impulses from the inflammation in the lung through the cervical sympathetics and the rami communicantes. Acute inflammation in the lung is expressed reflexly in motor; and chronic inflammation in trophic changes in these muscles the same as acute appendicitis is expressed in a motor reflex in the abdominal muscles. It is self-evident then, that the condition of these muscles offers useful data upon which to suspect both active and chronic intrathoracic inflammations and thus becomes of value in the diagnosis of pulmonary diseases.

Segmental Distribution of Nerves to the Somatic Muscles.—With our ever increasing interest in visceral neurology, we are not only led to study the functional changes in the internal viscera themselves, but also to determine the manner in which diseases of the internal viscera are reflected in the superficial tissues of the body. The key to this study lies in the fact that the body is made up of many different segments, and that each portion of the surface of the body which receives innervation from a given segment, is also bound, by reflex paths, to the various internal viscera which receive their nerve supply from the same segment.

In order to understand this we must bear in mind the developmental relationships between the cells in the spinal segments and the cells of the sympathetic system. Early in the course of development, the cells of the sympathetic nervous system push out from the segments of the cord, and now are found in peripheral ganglia. Each spinal nerve receives its nutrition from cells which lie within the cord itself. The cells which give origin to the sympathetic fibers going to the internal viscera and which originally came from a certain segment are no longer found in that segment but lie in ganglia outside of the cord. There are still, however, connector fibers which run between the cells of the segment in the cord and the cells in the sympathetic ganglia which have been pushed off from that segment, thus preserving the segmental relationship.

The reciprocal relationship between the viscera supplied from a given segment and the somatic structures supplied by the same segment must ever be borne in mind. Any stimulation, or irritation of the superficial structures of the body supplied by a spinal nerve, produces disturbances in the nerve cells of that segment of the cord from which it takes its origin, and this is transferred through the connector fibers to the cells in the corresponding sympathetic ganglion which give origin to the sympathetic fibers supplying the internal viscera. In case of inflammation, this irritation of the cells in the cord produces pathological change either temporary or permanent, which is reflected from the somatic structures to the internal viscera; and conversely, when internal viscera are inflamed, from the internal viscera into the somatic structures.

If only the paths of these afferent nerves can be accurately worked out we can then anticipate the localization of the resultant reflex action.

From this it can be seen that every inflammation of an internal organ produces an irritation in the cells of that segment of the cord which is in communication with it through the sympathetic afferent fibers and the connecting intercalated neurons; and that this may be expressed in the superficial tissues in sensory, motor, and trophic disturbances.

While there are some difficulties in the study of the exact innervation of the somatic structures, there is still greater difficulty in determining the intricate innervation of the internal viscera. At the same time, the whole scheme has been worked out in a manner to be sufficiently accurate for clinical work. There are still many clinical observations to be made and many physiological facts to be determined. The time is now ripe for clinicians and physiologists to cooperate and aid in the working out of these phenomena. For the study of the reflex motor and trophic phenomena which are seen when internal viscera are inflamed, I append the following table, prepared by Wichmann, and quoted by Bechterew[2] in which the somatic muscles, together with their segmental innervation, are shown.

Reference to this table will facilitate the understanding of the various reflexes which are described in the text, such as the reflex from the lungs to the muscles of respiration; that of diaphragmatic pleurisy; intercostal pleurisy; tuberculosis of the intestines; and tuberculosis of the kidney, each one of which will be described under its proper heading.

I. Cervical Roots.

C I-II	Deep neck muscles.
C I-III	Hyoid muscles.
C II (I-III)	Sternocleidomastoideus (Nervus accessorius Willisii).

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[2] Die Funktionen der Nervencentra, vol. i, Gustav Fischer, Jena, 1908.

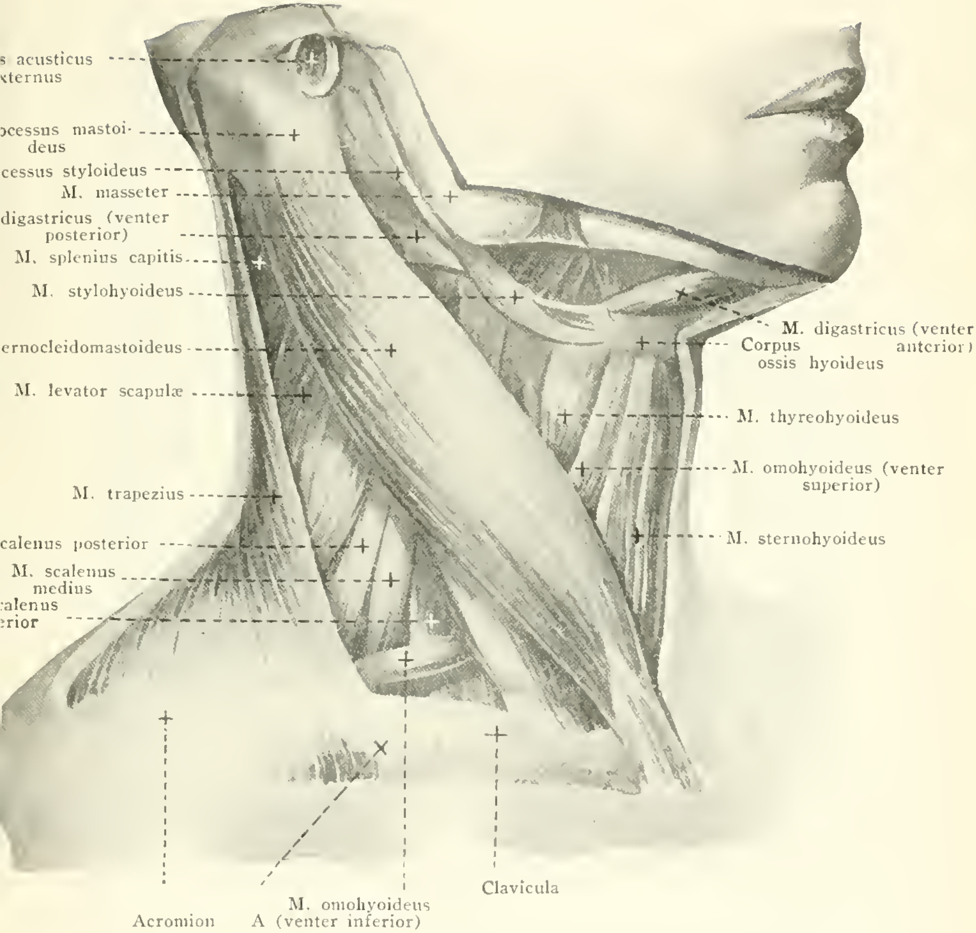

Fig. 61.—Showing muscles of the neck (side view). Sternocleidomastoideus and scaleni are of special diagnostic importance. (Spalteholtz.)

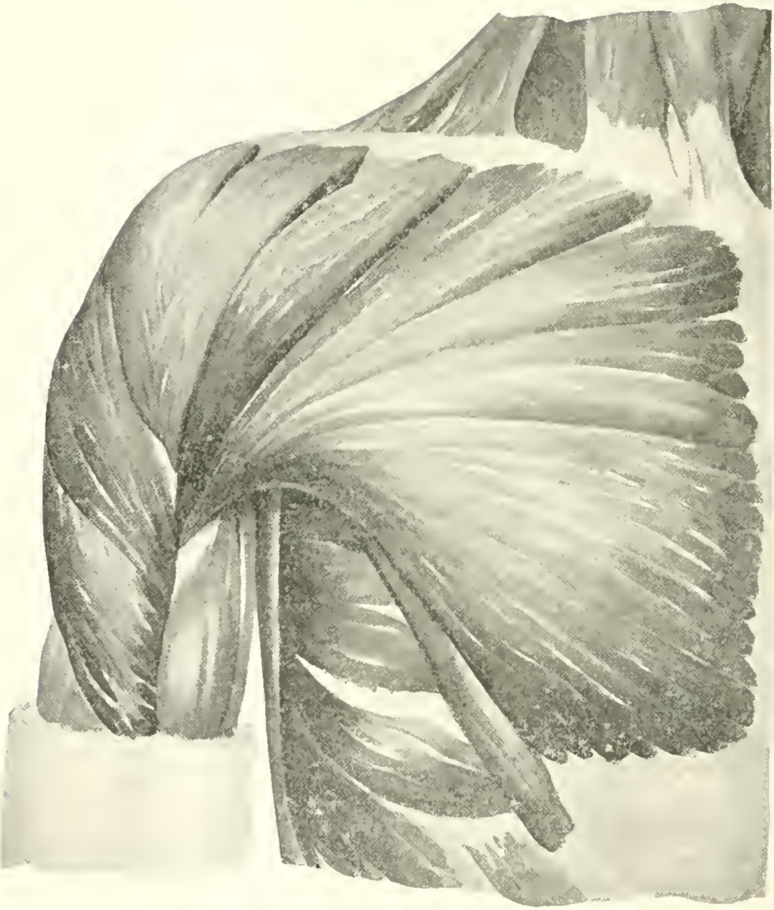

Fig. 62.—Showing the pectoralis. (Spalteholtz.)

C II-IV	Trapezius. According to Gowers, the middle and lower portion of this muscle is innervated through the lower cervical, and even the upper thoracic segments.
C II-VIII	Longus colli.
C III-VI	Scalenus anticus.
C III-VIII	Scalenus medius.
C IV (III)	Diaphragma (Nervus phrenicus), Levator scapulæ.
C IV-V	Rhomboidei.
C V (VI)	Supra- and infra-spinatus, Teres minor, Subscapularis, Subclavicus.
C V-VI	Biceps brachii, Brachialis (Nervus musculo-cutaneous).
C V-VIII	Scalenus posticus.
C VI (V)	Deltoideus (Nervus axillaris), Teres major, Pectoralis major (pars clavicularis), Coracobrachialis.
C VI-VII (V)	Serratus anticus major; Supinatores (Nervus radialis).
C VI-VIII	Latissimus dorsi.
C VII (VI)	Triceps brachii (According to Oppenheim the triceps center lies lower in the cord), Radialis externus, Abductor pollicis longus (Nervus radialis), Radialis internus, Pronator teres (Nervus medianus), Musculi thenaris (whose center, according to other observers, also lies lower in the cord).
C VII-VIII	Extensores digitorum, Ulnaris externus (Nervus radialis), Pectoralis major and minor.
C VIII (VII Th. I)	Flexores digitorum, Pronator quadratus, Palmaris longus (Nervus medianus).
C VIII-Th. I	Ulnaris internus (Nervus ulnaris). The small muscles of the hand, with the exception of the thumb, in so far as these are innervated through the median nerve, belong to a higher segment.

II. Thoracic Roots.

T I-IV	Serratus posticus superior.
T II-XI	Intercostals.
T V-XII	Rectus abdominis, Obliquus abdominis externus.
T VII-L I	Transversus abdominis, Obliquus abdominis internus.
T IX-XII	Serratus posticus inferior.

III. Lumbar Roots.

L I-II	Psoas, Cremaster.
L II-III	Iliacus.
L II-IV	Sartorius, Quadriceps femoris, Pectineus, Adductores femoris, Rectus internus.
L III-IV	Obturator externus.
L IV-V	Tensor fasciæ latæ (Nervus glutæus superior), Semimembranosus (Nervus ischiadicus), Tibialis anticus (Nervus peroneus).
L IV-S I	Glutei medius et minimus (Nervus gluteus superior) Gemelli, Quadratus femoris (Nervus ischiadicus).

L V	Semitendinosus (Nervus ischiadicus), Plantaris (Nervus tibialis), Extensores digitorum (Nervus peroneus).
L V-S II	Gluteus maximus (Nervus gluteus inferior), Biceps femoris (Nervus ischiadicus), Gastrocnemius, Soleus, Tibialis posticus, Flexores digitorum (Nervus tibialis).

IV. Sacral Roots.

S I	Obturator internus (Nervus ischiadicus).
S II	Pyriformis (Small muscles of the sole of the foot with the exception of the abductor pollicis and Extensor digitorum brevis, which belong to the 5th Lumbar and 1st Sacral.
S III-V	Muscles of generative organs.

Influence of Muscles and Soft Tissues on Physical Findings.—Figs. 61, 62, 63, and 64, pages 330 and 332, show the most important neck and chest muscles. It is necessary for one to familiarize himself with them in order to appreciate the influence which motor and trophic changes in them exert upon palpation, percussion and auscultation.

The thickness of the soft tissues covering the lung varies over different portions of the chest wall. Over the pectoral, scapular and interscapular regions it may be one or two centimeters in individuals with slight musculature or four or five centimeters in those in whom these tissues are well developed. The apex of the lung, posteriorly, where covered by the trapezius, lies from four or five centimeters to six or eight centimeters below the surface. The influence of this varying thickness of soft tissues upon the data obtained in physical examination is extremely important.

It can readily be understood that such musculature when of increased tone (spasm) will afford different data from what it would when normal. This can be appreciated particularly on percussion. It can also be seen that, when a degenerative process affects these soft tissues (skin, subcutaneous tissues and muscles all degenerate), as happens regularly in chronic tuberculosis, resulting in a loss of substance at times up to nearly half the total amount, this, too, greatly modifies the data obtained upon physical examination. The importance of the tone of these soft structures may be inferred from their thickness as shown in Figs. 65 *A*, *B*, and *C*, page 334. This degeneration is all the more important because it is regional in character and usually affects one side

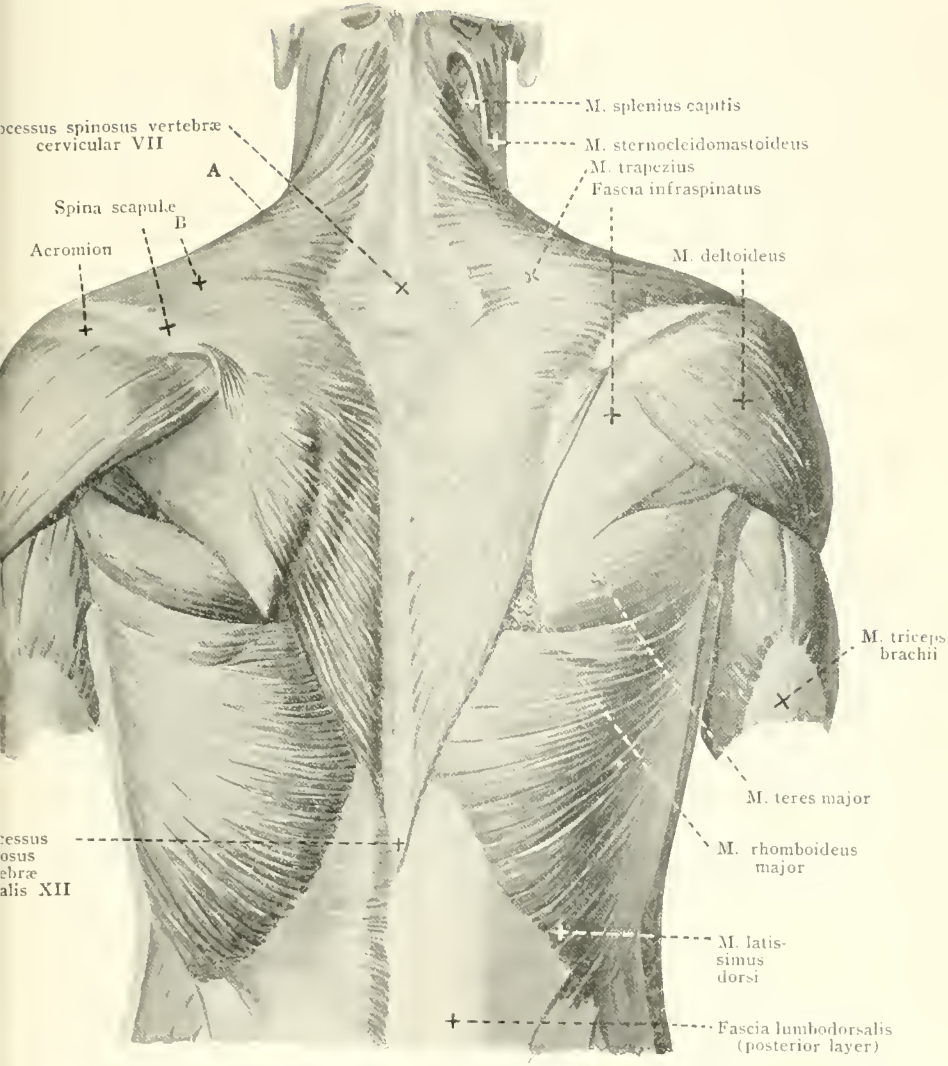

Fig. 63.—Showing superficial muscles of the neck and back (posterior view). Trapezius of great diagnostic importance. *A* and *B*, portions of trapezius which show spasm and degeneration best. (Spalteholtz.)

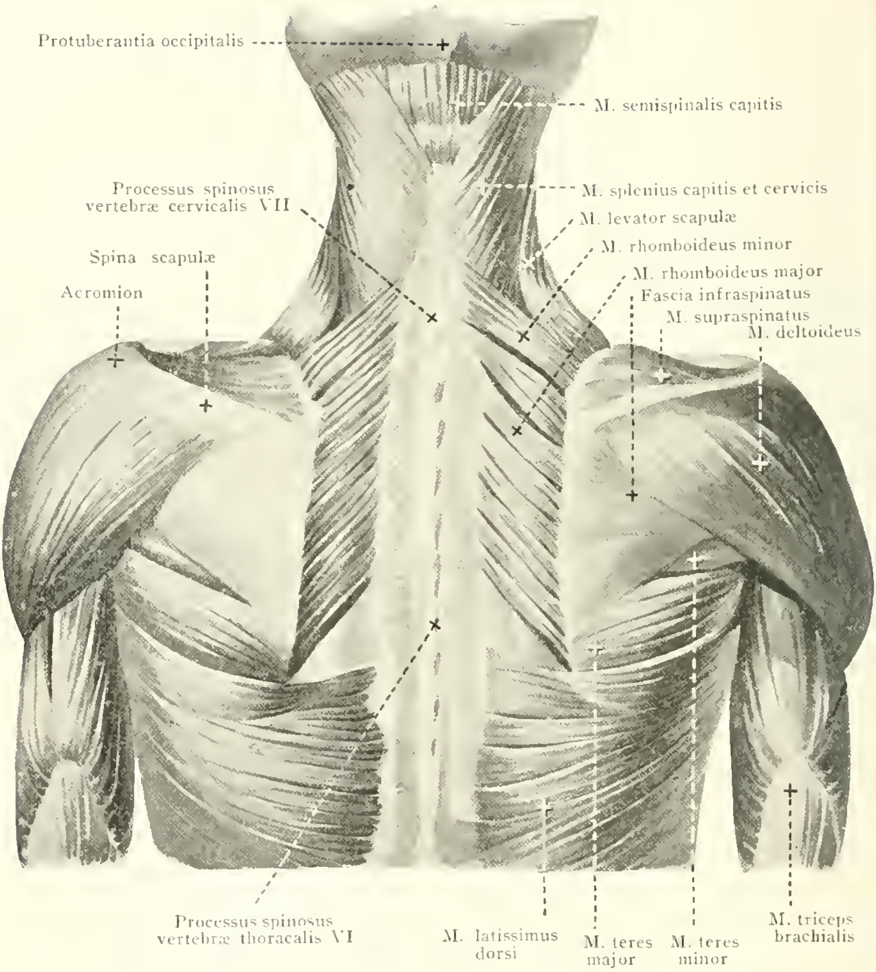

Fig. 64.—Showing the second layer of muscles of the back. Levator anguli scapulæ and rhomboidei of special diagnostic importance. (Spalteholtz.)

(the side of the oldest lesion) far more than the other. The effect on the shoulder girdle is shown schematically in Fig. 90, page 422, and clinically in Figs. 95, *A* and *B*, page 466.

Common Occupational Changes in the Soft Tissues of the Thorax.—Inasmuch as the condition of the soft structures covering the thorax offers important diagnostic signs in pulmonary tuberculosis, it is necessary that we familiarize ourselves with those things which cause a departure from the normal. The discussion of the pathological variations from the normal will be found elsewhere in these pages. In my study of the reflex spasm and degeneration of the muscles, and the degeneration of other soft tissues, caused by inflammatory processes within the lung, the conditions which have caused me most trouble have been those due to changes which result from occupational influences.

Right-handedness is almost universal. The fact, however, that there are a large number of people who are left-handed, gives us an opportunity to draw fairly accurate conclusions from the changes which come from more or less constant use of one hand in preference to the other. We have found that people who use their right hand most, not only in muscular acts, but in carrying objects as well, have a lowering of the shoulder on that side, as compared with the other shoulder. In persons who are left-handed, we find this lowering of the shoulder on the left side. This indicates that there are certain changes which take place in the supports of the shoulder girdle on account of constant use. Examination shows this to be a lengthening; and, apparently, at least, a degeneration of the shoulder muscles, which is particularly emphasized in the trapezius and levator anguli scapulæ. There is also an apparent degeneration or thinning noted in the pectoralis on the side of the arm used most. This lengthening and degeneration of the muscles is almost universal in the human family, except in those individuals who do heavy work with these muscles. Such individuals very often show a marked hypertrophy which almost, and, at times, wholly, overcomes any stretching or thinning of the muscles that would otherwise be evident.

Normal Variation in Physical Types and Their Visceral Function.—An error in both physical and functional diagnosis arises from attempting to consider all individuals from a given standard. Such is impossible. Among other things, individuals differ in general contour of the body, in size, in weight, in the amount of flesh, in the shape of the thorax, in the shape of the abdomen, in muscular strength, and nerve stability. Of necessity, the organs must differ in form and functional capacity. We cannot have the same shaped lungs, the same shaped liver, the same position of the kidneys, and the same position of the intestines in a long slender body that we have in a short thick body. Neither can we have the same degree of functional capacity and functional activity in all individuals.

There has been too much of a tendency to try to measure all individuals by the same standard. In examination of the chest a low diaphragm in one patient would not be a low diaphragm in another. The heart must assume an entirely different position in a long chest from what it does in a short chest. We cannot judge the position of the abdominal viscera with relation to the umbilicus in all individuals. The examiner must bear in mind, therefore, that the organs must fit in according to the size and shape of the cavity in which they are placed. In this way only can he learn departures from the normal. Clinicians must also bear in mind that there is no normal functional activity for all individuals.

In discussing the functional activity of the gastrointestinal tract, observers tell us that the stomach should be empty in six hours; consequently, we fix in our mind that anything short of six hours is too rapid, and anything later than six hours is too slow. Such is erroneous. An individual with a stomach in a high state of functional activity and strong musculature may empty this organ in less than six hours, without being looked upon as having hypermotility. On the other hand, a person with a less degree of functional activity with less muscular strength, might require longer than six hours and still not suffer from hypomotility.

Our conception of the normal must be altered to suit the physical and functional capacity of the individual.

To simplify matters, however, individuals may be classified in certain groups in which the shape, relationship, and functional capacity of organs may be inferred from the physical form and degree of nerve stability of the individual.

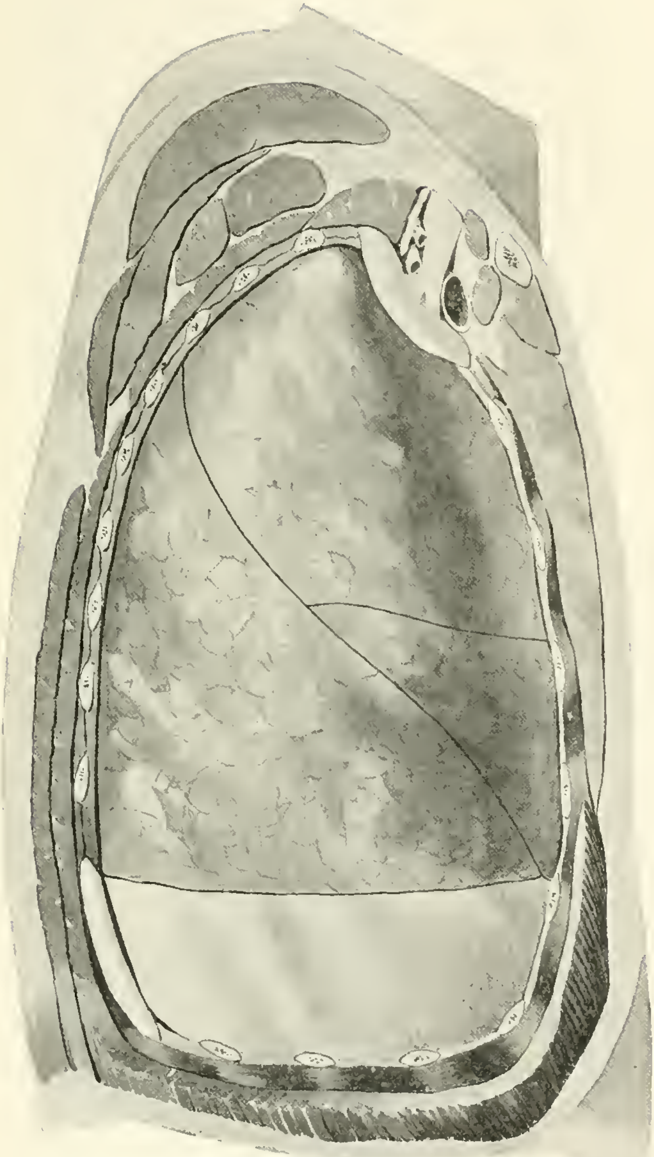

Fig. 65*A*.—Sagittal section through the body showing the thickness of the soft structures covering the apex from which may be inferred the importance of the increased tone (spasm) or degeneration upon the findings on palpation, percussion, and auscultation. Anterior view. (Corning.)

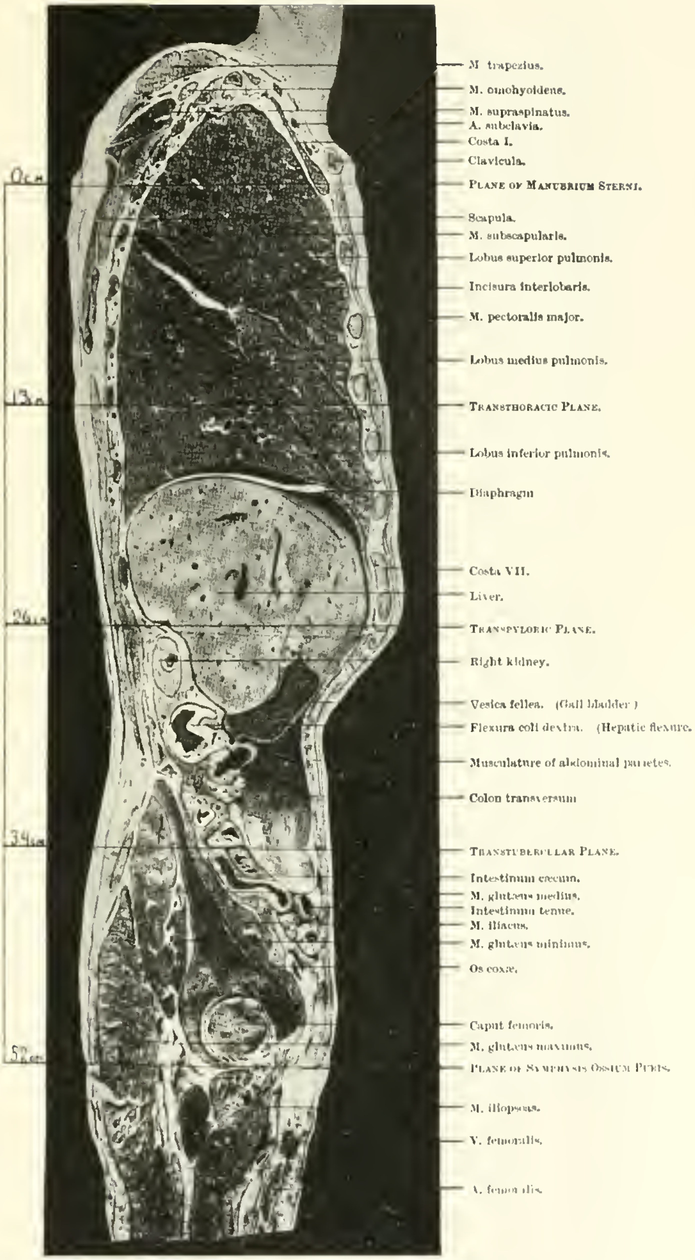

Fig. 65*B*.—Section through body 6 cm. to the right of the median plane, viewed from the right. Showing the importance of the soft tissues as influencing physical examination of different areas of the chest. (Berry.)

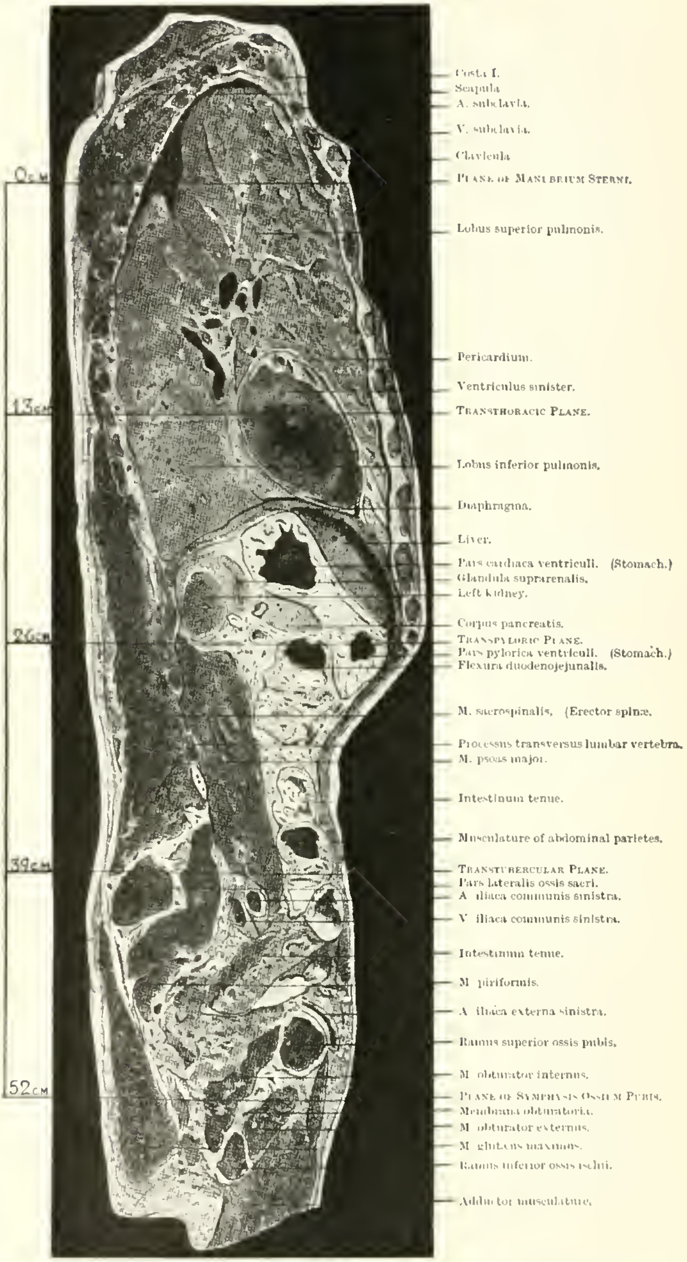

Fig. 65*C*.—Section through body 6 cm. to the left of the median plane viewed from the right. Showing the importance of the soft tissues as influencing physical examination of different areas of the chest. (Berry.)

These facts have been particularly emphasized by the splendid original work of Dr. R. Walter Mills, of St. Louis.[3] Owing to the extremely great importance of this subject for all practitioners of internal medicine, I wish to append the following original manuscript prepared especially for this monograph by him.

THE RELATION OF VISCERAL FORM, TOPOGRAPHY AND FUNCTION TO THE GENERAL PHYSIQUE, WITH A CLASSIFICATION OF TYPES.

By R. Walter Mills, M.D.

If by means of the x-ray any considerable number of subjects are studied with regard to the topography of their thoracic and abdominal viscera the factor of individual variation becomes so evident as to make the futility of any single topographical standard exceedingly apparent. The massive body of a heavy powerful man houses a heart so different in outline from that of a slender woman of less than half his height, as to make them appear almost as different organs. In the same way the general outline of the lung fields, the silhouette of the diaphragm, and the form and position of the abdominal viscera vary widely in extremes.

If a series of subjects are so studied as to graphically and accurately reproduce the visceral outlines in that of the bodily figure, certain constancies as to relationship between visceral forms and position and the general physique become apparent; for instance, that a certain type of powerful massive individual always houses a heart and lung fields of characteristic outline, and that such are never normally found in other bodily types.

If the above conception be true, there must be controlling factors governing such relationship. It would seem that there are such factors. First and chiefly the essential individual skeletal architecture. The subject of massive osseous plan presents a thoracic cage capacious, deep in its anteroposterior dimensions, wide in its lower lateral, and relatively short as compared to other types in its longitudinal axis. This latter is influenced by the arrangement of the abdominal regional capacities as such a subject has a high digestive plant in turn due to a relatively small pelvic capacity and considerable space occupying abdominal fat. A chest of such fixed regional capacities can only house lungs of certain form.

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[3] Observations on Duodenal Ulcer with Special Reference to Its X-Ray Diagnosis, Interstate Medical Journal, vol. xxiii, no. 4, 1916.

The second determining factor in the relationship of visceral topography to bodily habitus is variation in the muscular tonus of different persons. By tonus we understand that inherent resilience possessed in varying degrees by all living muscular tissue. The muscular tonus of the alimentary viscera is a chief factor in giving them their form; it seems highly probable that the form of the heart in like manner may be influenced by the tonus of its musculature. The tonus of the skeletal muscles and intimately associated with it their degree of muscular strength contributes to visceral form by influencing the form of the thoracic and abdominal cavities. A skeletal musculature of a high degree of tonus and strength makes for integrity of the general static poise; hence there is less tendency to spinal curvature, figure collapse, and postural abnormalities. Again, a well-developed belly musculature is a considerable factor in influencing the form of the abdominal and secondarily, the thoracic viscera.

The degree of nutrition plays a role in determining the form of the viscera; where poor, the abdominal viscera are lacking a considerable support in the way of intra-abdominal fat, and occupy a lower position than in well-nourished subjects because the pelvis is more roomy. In turn the upper abdomen being of lesser content is smaller and the form of lung fields and heart consequently influenced being of longer longitudinal dimensions. The degree of nutrition furthermore modifies visceral form in overnourished subjects through its influence on static conditions and the form of the abdomen. A heavy pendant abdomen results in a somewhat lower position of the abdominal viscera than is normal for that particular subject judged by the general skeletal type with consequent influence upon the form of the thoracic viscera.

The physiological needs of the individual influence the form of the viscera. In one of robust and heavy figure, the heart must serve greater demands and is consequently larger and of a form resulting from a heavier cardiac muscular development. The abdominal viscera having to accommodate and digest a larger amount of food proportional to the metabolic needs of the individual are of a form favorable to a more rapid alimentary motility than occurs in slender persons.

All factors influencing bodily and consequently visceral form act in varying degrees in the individual case and frequently attain an extraordinary complexity in the determination of the final visceral type and arrangement. On the other hand, one factor may be so dominant as to permit of but little change from the essential plan even though all others be greatly modified; for example, a subject may be primarily of so massive and powerful a physique that no alteration in those other factors usually influencing visceral form serves to change the plan. Muscular tonus and strength may be decreased through debility, great loss of weight occur, and still not greatly alter that essential visceral type and arrangement characteristic of such physique. In that other extreme of habitus, the congenital type having normally a pelvic digestive plant, long gracile thorax, pendant heart and poor muscular tonus, no increase in fat or artificially developed musculature, no improvement in general well being serves to alter the essential stigmata of such type. It is in intermediate types of physique that the balance between the various factors resulting in the individual visceral topography are most delicately balanced, so that a marked loss in the integrity of one factor often results in distinct change in form and position. A man may be of average build, strength and nutrition; in him a marked deficiency in alimentary tonus may result in distinctly different alimentary outlines from what his general physique would lead us to anticipate.

From what has been said, it may be gathered that the position is here taken that there is no single type of visceral form or position that may be considered normal, and that all others not corresponding thereto are abnormal. We may no more elect one normal visceral standard than we may elect one individual's physique as a standard for all persons. Rather we must hold that innumerable types of physique, visceral form and topography occur as a rule representing normal conditions and that an abnormal status only exists when there is a gross departure from the essential bodily plan of that given individual as judged by standards established by studies of large numbers of subjects of similar type.

In addition to great variation in the form and position of the viscera any extended observations of different subjects will show an equally wide variation in certain physiological processes; perhaps the most striking being difference in the degree of visceral tonus. Here too a relationship will be found to exist between the general bodily habitus and the degree of tonus. In those of a certain type of powerful physique, the degree of visceral tonus will be found to be great, while in those of an opposite type—asthenics—"congenital enteroptotics," it is poorest.

There are before us certain considerations: First, an infinite variation in bodily physique, visceral forms and in certain physiological processes. The presence in this great series of variations of certain dominant types of physique and visceral topography also well-marked degrees of certain physiological processes. A constancy of relationship between certain types of physique, certain visceral forms and degrees of physiological manifestations is also evident. Second, a very evident need for more accurate standards by which departures from normal conditions whether morphological, topographical or physiological may be judged in the given case. This for diagnostic ends and for purposes of study. A need that it seems perfectly evident can not be subserved by selecting one type as normal and considering all other types—consequently a majority of all persons—as abnormal. On the other hand it is obviously impracticable to have an individual, though in many ways ideal, standard of anatomy and physiology for each person. Third, the possibility that failing both a single standard for all and an individual standard for each one, we may through a classification of types as to physique and their parallel visceral peculiarities best serve our ends. This we believe is practical and while a difficult matter on account of the amazing complexity in which different characteristics are often present in the same individual, offers us a basis for further study that is more satisfactory and hopeful than our former one type standard.

An investigation of hundreds of individuals in whom the visceral plan has been graphically recorded in its relation to bodily contour, has shown that in two extremes the relationship of habitus to visceral form, position, tonus and motility, is a constant. Since the question of chest topography is chiefly of interest here, special attention will be given it though it must be emphasized that both thoracic and abdominal topographies and forms are commonly characteristic of certain types of habitus and consequently they bear to each other a definite relationship. In the following descriptions of topographical relations the individual is considered as in the standing position with the stomach filled to the same degree as after the standard bismuth meal. The first of these two dominant types in which the general physical and visceral peculiarities are dominantly characteristic has been classified as hypersthenic (Fig. 66). Subjects of this type are of the most powerful physique and usually very heavy. The body framework is exceedingly massive, the thorax deep anteroposteriorly, wide in its lower lateral diameters and short longitudinally. The costal arch is so obtuse as to be sometimes almost a straight line, giving the figure a peculiar gorilla-like appearance. The pelvis is comparatively small. Corresponding to the form of the thorax, the lung fields are broad at the base, the general direction of their lower borders approaching the horizontal. The lung fields contract markedly from base to apex giving the combined lung fields the form of a truncate pyramid. The apices are small and extend but little above the clavicles, less so than in any other type. The form of the heart being very characteristic in the hypersthenic, influences the outline of the lungs, especially the left. The heart is peculiar in that it occupies an almost transverse position. It seems largely below the silhouette of the diaphragm, giving an appearance of being half submerged. Often the outline of the left ventricle is almost a continuation of the lower border of the left lung field. The outline of the aortic arcus as silhouetted by the x-ray is short and broad, and the shadow of its apex projects less to the left than in less sthenic types. As viewed from the front the lung area appears smaller than in other types, only apparently so as the lungs are much deeper than in persons of slender habit and their volume is consequently in keeping with the size and weight of the body. Owing to the short thorax and the long abdomen characteristic of the hypersthenic habitus one of the most peculiar features of the type is the very high digestive plant, the stomach being almost thoracic and the intestines especially high in position. The visceral tonus is invariably of the highest degree, stomach, small intestine, and colon being equally striking as to their hypertonicity. The stomach is of Schlesinger's hypertonic form or Holzknecht's bull-horn type. The pylorus is the lowest or nearly the lowest part of the stomach. The small intestine is high in the abdomen and equally characteristic as to its hypertonicity which is shown especially by the narrow worm-like outline of the loops of ileum in contradistinction to the broad more or less regular shadows of the ileum in subjects of poor visceral tonus. The colon is very high in position, the cecum being well above the iliac basin, even with the patient standing. The transverse colon is horizontal. The descending colon is long owing to the high position of the intestines and more nearly straight in its entirety than in other types. Visceral hypertonicity characteristic of hypersthenics is possibly most strikingly shown in the multiplicity of the colonic haustræ and their sharp and deep demarcation. Alimentary motility is faster in the hypersthenic than in any other type. The contrast meal pours from the stomach immediately on ingestion. Small intestinal and colonic motilities are commensurably rapid, the contrast substance being frequently passed per rectum within twelve hours. Such subjects characteristically defecate two or more times in the twenty-four hours. The pure type is rare. I have seen less than a dozen in x-ray type studies of some two thousand subjects—I have never seen a woman of pure hypersthenic habitus though a few have been observed that approached it. Such women are far more masculine in their general bodily physical characteristics than are many men.

In order to best appreciate variations in type both of physique and topography, it will be best to consider next that habitus which is the antithesis of the hypersthenic, the second dominant type the asthenic (Fig. 67). The asthenic type occurs at the other end of the scale in a series of types representing gradations between extremes. Stiller immortalized himself by his conception of this type, his "Asthenia universalis congenita," though his work has had to await the x-ray for its fullest appreciation. Persons of this type are most commonly women of frail slender build having a delicate bony structure, feeble musculature, and but little body fat. The thorax is long and gracile, the intercostal angle is narrow and the ensiform absent or rudimentary. The pelvis is flat and capacious. The most striking and essential characteristic of the asthenic is the marked disproportion between the great capacity of the pelvis and the limited capacity of the upper abdomen and lower thorax, this of necessity determining a pelvic digestive plant and a thoracic topography corresponding thereto in its disproportionately long longitudinal dimensions as compared to its short lateral distances. How great this disproportion between the pelvic capacity and that of the upper abdomen may be appreciated by considering other factors. The pelvis of a woman of pure asthenic type is often far broader and more capacious than that of a woman approaching the hypersthenic type, this though the lower thorax of the latter is twice the capacity of that of the former and the body weight frequently twice as much. The form of the lung fields is markedly influenced by the asthenic habitus; they are relatively broad in their upper zones and narrow in their lower, in contradistinction to those of the hypersthenic. The diaphragm slopes sharply downward to each side though very frequently this is less marked owing to general static changes resulting in a degree of figure collapse. The lung apices are large and extend well above the clavicles. The lung fields appear large for the individual probably because anteroposteriorly shallow and on account of the meager fat and muscular clothing of the body frame. The heart is of that peculiar form described as drop heart being narrow and pendant. It is median in position its long axis being in that of the longitudinal axis of the body. In general its position is well above the diaphragm. The aortic outline is long narrow and above is deflected to the left as a club-shaped terminal. In keeping with the relatively great capacity of the lower abdomen, the digestive plant is low in position. The stomach is largely pelvic as are the ileum and colon. The form of the various portions of the digestive tube is characteristic and some portions bear hardly a resemblance to the similar structures of the hypersthenic. The stomach appears as a pendant sac and is of that peculiar form designated by Schlesinger as atonic.

The ileum is capacious and the colon is characterized by its large cecum and the coarseness of its haustration. Equally striking and constant with the differences in position and form of the viscera in the asthenic and hypersthenic is the difference in the

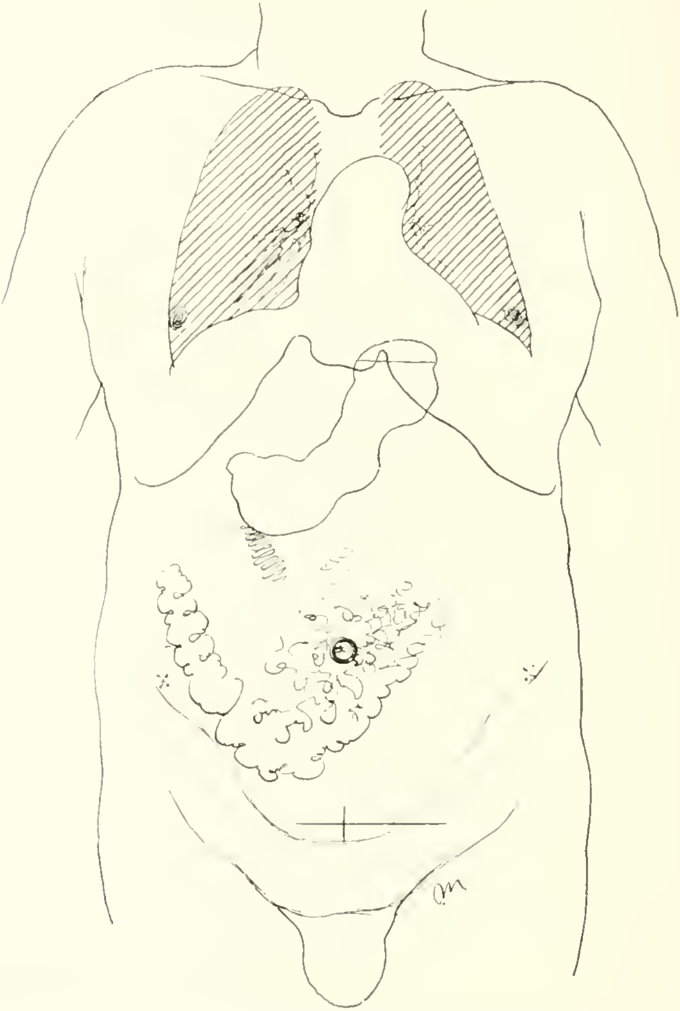

Fig. 66.—The hypersthenic habitus, a dominant type. Essential characteristics are massive figure, short deep capacious thorax, very obtuse intercostal angle, with wide well-developed ensiform. Lung fields very wide at base and narrowing rapidly to their apices. A heart whose longitudinal axis is almost horizontal. A long abdomen relatively more capacious in its upper zones and housing a digestive plant of very high position. The highest degree of visceral tonus and the most rapid alimentary motility of any type are other characteristics. (Mills.)

degree of alimentary tonus. The essential tonal attribute of the asthenic is atony. The parts of the alimentary tube in the asthenic are never endowed with sufficient muscular tonus to support their contents. The contractility of the sphincters is also poor as shown by the ease with which the pylorus may be

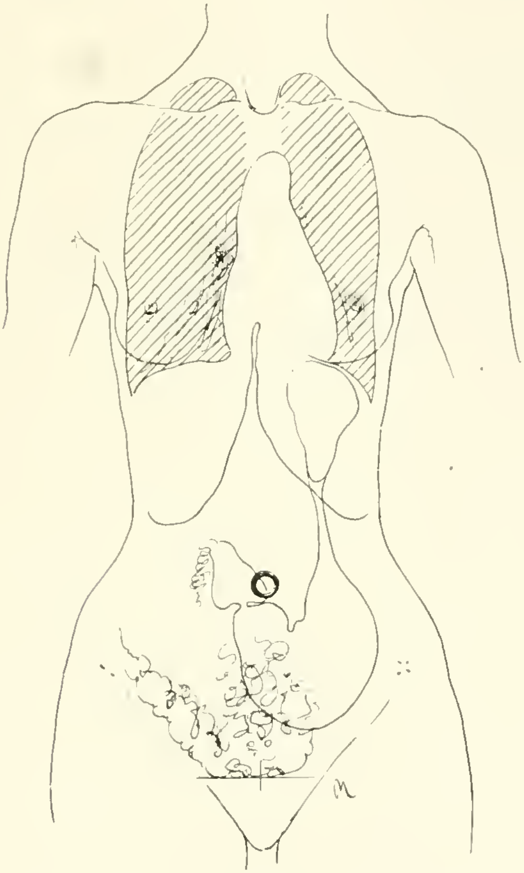

Fig. 67.—The asthenic habitus, a dominant type. Essential characteristics are frail, slender figure, a great disproportion between the capacious pelvis and wide hips and narrow upper abdomen and lower thorax, a very narrow intercostal angle with no or only a rudimentary ensiform is constant. The lung fields are relatively narrow in their lower zones and wide in their upper. The diaphragm slopes sharply downward to both sides from the median line. The heart is central in position, its long axis being approximately in that of the body median line. It is of characteristic pendant form and its shadow is but little covered by the silhouette of the diaphragm. Stomach and intestines are very low in position, conforming to the regional capacities of the abdomen. The form of the stomach and colon is very characteristic. The degree of alimentary tonus is the poorest and gastro-intestinal motility the slowest of any type. (Mills.)

overcome by manual manipulation as shown by the fluoroscope and the poor tonus of the rectal sphincter. Alimentary motility is slower in the asthenic than in any other type. The stomach not infrequently does not completely empty within six hours after a standard bismuth meal, this without organic cause for such. The motility of small and large intestine is commensurably slow. The comparison of asthenic and hypersthenic might be continued to show an antithesis in almost every physical physiological and psychical attribute.

In the assumption of such an idea as of a multiplicity of normal anatomical and physiological types one may ask on what basis such a conception rests, a conception opposing the generally accepted idea of a single normal type with variations from such, for instance, low position of the viscera representing acquired pathological conditions, an idea that has been accepted since the days of Glenard and emphasized by the writings of Ewald, Wolkow and even Stiller, who while accurately describing the type designated by him the asthenic, held that such type is peculiar in that it predisposed to visceroptosis.

Of any single species of organism man shows the widest variaion in his visible characteristics. This owing to his wide geographic distribution, to great difference in his manner of living and most to the advent of reason as a modifier of those natural evolutionary processes that have resulted in the creation of fixed species among the lower animals who vary but little from their species type and within limited bounds. There is frequently greater physical variation among the children of the same parents than in different species among the lower animals. One has but to suggest the difference in faces and hands or of the ratio of body height to weight. Man is not a species of fixed physical characteristics varying within narrow limits. It does not seem reasonable that the viscera housed in bodies varying so widely must be of one type as to form and arrangement to be considered normal. Again, certain bodily structures whose form could not possibly be modified by any acquired characteristic such as visceroptosis, are as characteristic of certain types associated with low position of stomach and intestine as are other bodily peculiarities that might be interpreted as the result of such ptosis. For instance the form and relative capacity of the pelvis in the asthenic type could not possibly be changed by any degree of splanchnoptosis, yet the proportions and structure of the pelvis are absolutely typical of such habitus. It is always possible under usual conditions to anticipate the peculiar form and low position of the stomach of an asthenic from a radiograph of his pelvis alone.

Further it is difficult to reconcile ourselves to the idea of a condition being essentially pathologic that exists in so great a proportion of healthy subjects. It is true that asthenics are of an inferior type; that they are not strong physically; that they are nervously unstable, fatigable and prone to digestive disorders. On the other hand, every walk of life is crowded with those of this type who never had a digestive disturbance and who frequently show a surprising endurance and capacity for work. It is not meant to imply that ptosis of the viscera does not occur, but the position is taken that before such is assumed there must be a departure from that abdominal topography to which the individual is entitled on the basis of his general structural type.

On the same basis we may assume with equal propriety and utilize diagnostically an elevation of the viscera when the position of such is higher than the type standard for that particular person. A scirrhus carcinoma of the stomach in an asthenic results in a stomach of higher position than we should consider normal on the basis of physique. On the other hand if a benign pyloric stenosis with resulting atony occurs in a hypersthenic subject the stomach may be low for that particular type of individual though still well above the umbilicus.

One of the strongest arguments in favor of a type anatomy is that certain persons, essentially asthenic yet having none of those physical peculiarities supposed to cause ptosis still have abdominal viscera of low position and characteristic asthenic form. A number of studies of women have been made who present the essential structural characteristics of the asthenic yet who weigh as much as one hundred and eighty pounds, are fairly muscular, and of good static poise. This visceral topography is purely asthenic, the stomach and intestines being low in position, of a relatively poor degree of tonus, and of that characteristic form typical of the "congenital enteroptotic."

It is not contended that the relationship between bodily habitus and visceral form, position, tonus and motility is always constant.

Not infrequent variations occur but it is held that in extremes of type this relationship is positive and in intermediate types it holds to a degree that makes a type anatomy and physiology the most hopeful possibility of escape from the chaos that has resulted from our present one type standard.

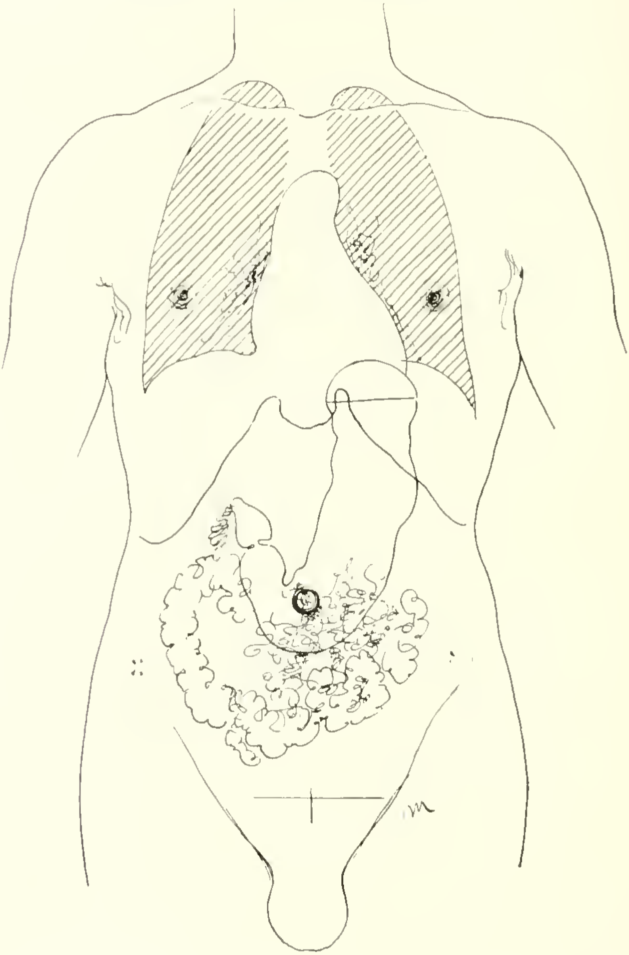

Fig. 68.—The sthenic habitus, a major type. The characteristics of this type are very similar to those of the hypersthenic, but differ in that they are all less marked than in that type (Fig. 66). Thus the general physique, while as a rule heavy and powerful, lacks the peculiar massiveness of the hypersthenic. In the sthenic the thorax is short and wide, the intercostal angle of about ninety degrees. The lung fields are relatively wide in their lower zones. The longitudinal diameter of the heart is somewhat transverse. The abdomen is rather long the alimentary viscera high in position. Gastrointestinal motility is fast and stomach and intestinal tonus of high degree. (Mills.)

The principle of type topography has been illustrated by the description of the two most widely varying types, the hypersthenic and asthenic. Between these two occur an infinite variety of physical forms and visceral plans. Among these are two types

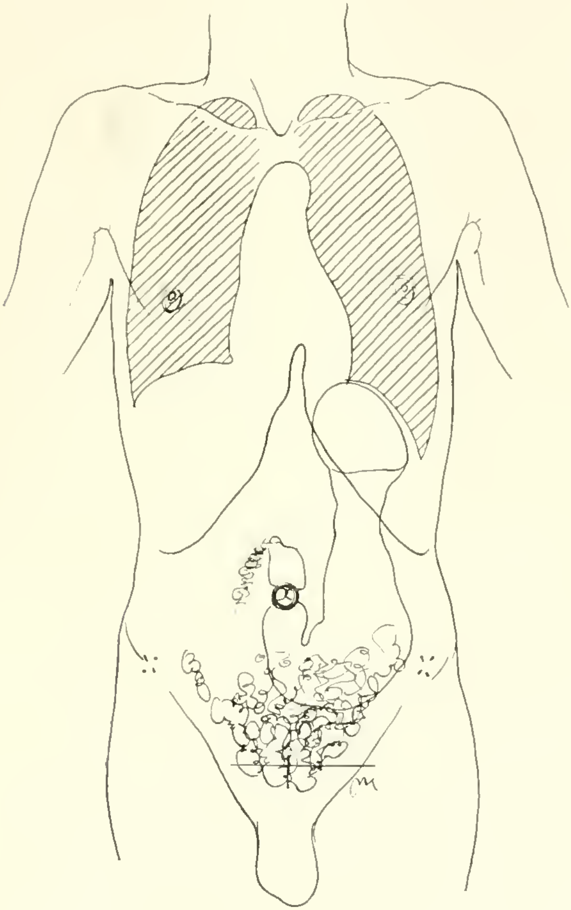

Fig. 69.—The hyposthenic habitus, a major type. Essentially an intermediate habitus, numerically common. It is the most difficult type to classify, as all characteristics approach a mean. In general, the structural characteristics are sthenic, while the visceral arrangement is more that of the asthenic. Hyposthenics also most frequently resemble the asthenic in their lack of robustness. The thorax is moderately long the intercostal angle narrow, usually about forty degrees, with the ensiform rudimentary or lacking. The lung fields are intermediate in their general proportion between those of the sthenic and asthenic types. The heart resembles that of the asthenic. Frequently it is of quite pendant form. The stomach is moderately low in position and the intestines occupy a relative position. Visceral tonus is rather poor on the whole though of higher degree than in the asthenic. Alimentary motility is of average rate. (Mills.)

that seem constant enough to afford a basis of type classification. The first is the sthenic (Fig. 68) and the second the hyposthenic (Fig. 69). As the names imply, they are types in which sthenic characteristics are dominant though differing in their degree. Our classification may be now stated as a division into four major types arranged in gradations: hypersthenic (Fig.

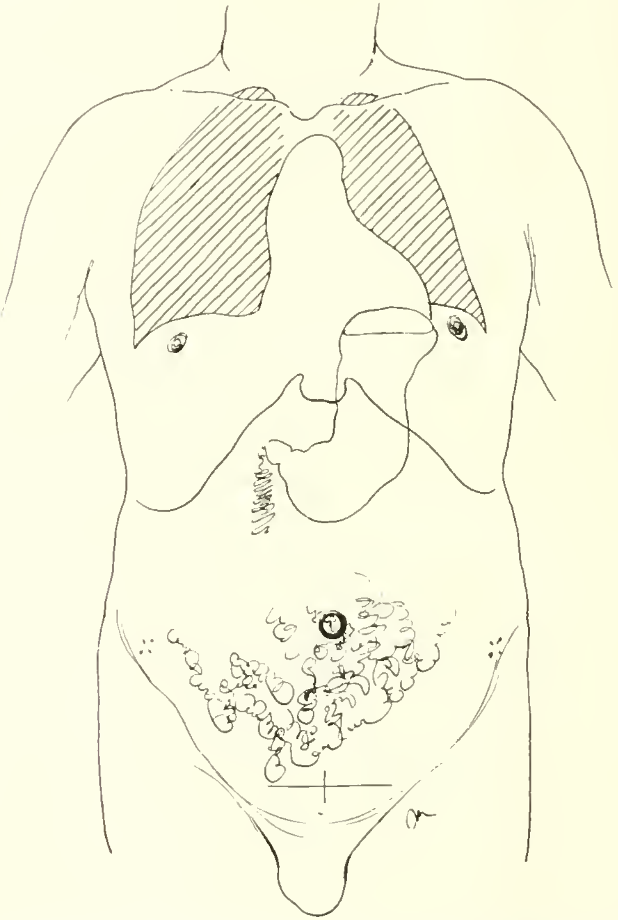

Fig. 70.—The hypersthenic to sthenic habitus, a sub-type. In this habitus physical and visceral characteristics are essentially hypersthenic (Fig. 66) though not as markedly so as in that type but tend somewhat to the next lower form, the sthenic (Fig. 68). Thus the thorax has not quite the depth of the hypersthenic and the intercostal angle is less obtuse. The stomach is not quite so extreme in form and position; other characteristics are relatively modified. (Mills.)

66), sthenic (Fig. 68), hyposthenic (Fig. 69), and asthenic (Fig. 67). Variation in the physique and visceral topography of different subjects is so great that this classification is not sufficiently elastic to lend itself to practical use. We must have finer subdivision of these types. To meet this requirement every subject is primarily classified as of that one of the above four divisions that his essential bodily peculiarities assign him. Should his general habitus be purely that of one of these types, no

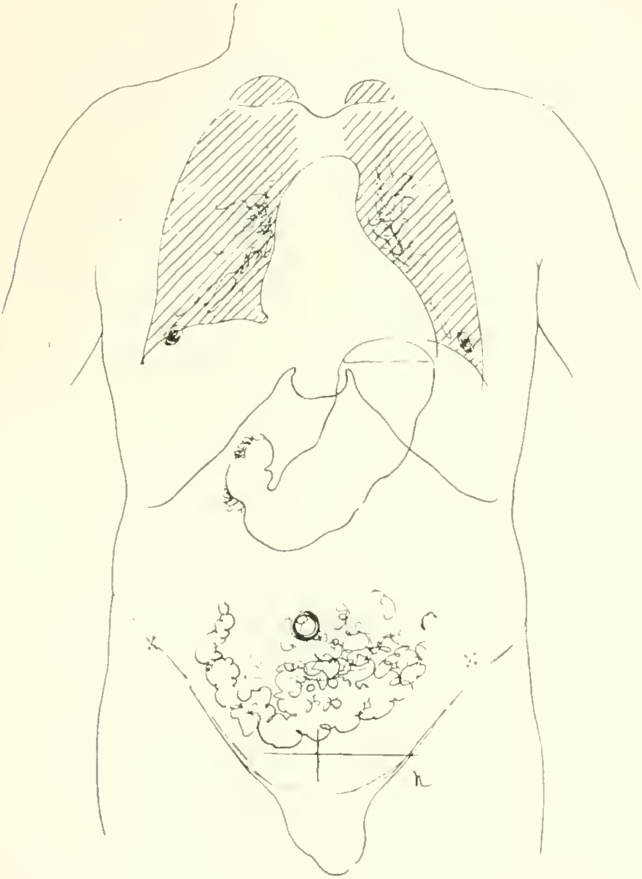

Fig. 71.—Sthenic to hypersthenic habitus, a sub-type. Here the essential characteristics are those of the sthenic (Fig. 68) but somewhat more suggestive of the hypersthenic (Fig. 66) than is the case in the pure sthenic. The thorax is a little shorter, the intercostal angle somewhat wider and the whole figure more massive than in the sthenic. Visceral form, position, and other peculiarities are similarly modified. (Mills.)

further classification is necessary. If however he be essentially of one of them yet present other characteristics suggesting the group above or below him, he is classified as primarily of that one of the four major types that he most resembles yet tending to another. We thus have six sub-types (Figs. 70, 71, 72, 73, 74, and 75). An individual may be essentially sthenic yet tend to

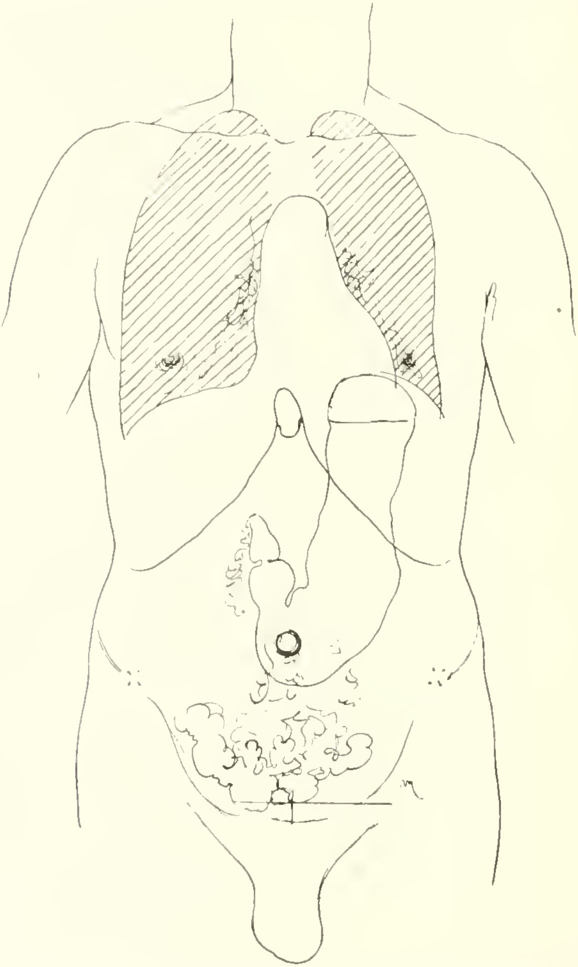

Fig. 72.—Sthenic to hyposthenic habitus, a sub-type. While the general structure is here sthenic (Fig. 68) there is a tendency to the hyposthenic (Fig. 69). This is shown by a less marked robustness, a longer thorax and shorter abdomen than in the pure sthenic. The visceral topography and other peculiarities are proportionately intermediate. (Mills.)

the hypersthenic in which instance he is classified as "sthenic to hypersthenic" (Fig. 71), or he may be dominantly a sthenic yet tend down the scale to the hyposthenic. He would then be classified as sthenic to hyposthenic (Fig. 72). This classification is partly the result of an effort to build, as is fitting, on certain

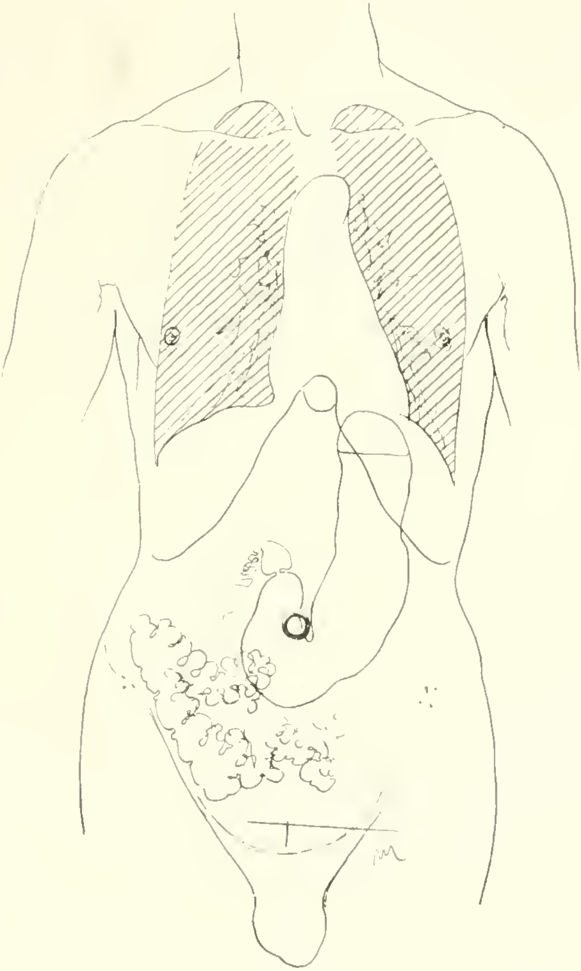

Fig. 73.—The hyposthenic to sthenic habitus, a sub-type. In this sub-form the characteristics of the hyposthenic (Fig. 69) are dominant though the figure and alimentary peculiarities are somewhat more sthenic (Fig. 68) than in the pure hyposthenic. Thus the intercostal angle is wider, the position of the stomach higher and its tonus more marked. (Mills.)

work that has already been recognized, namely Stiller's description of his asthenic and sthenic types and Schlesinger's classification of stomach forms into hypertonic, orthotonic, hypotonic, and atonic. This latter classification lends itself as a parallel to the above classification. Schlesinger's hypertonic stomach is

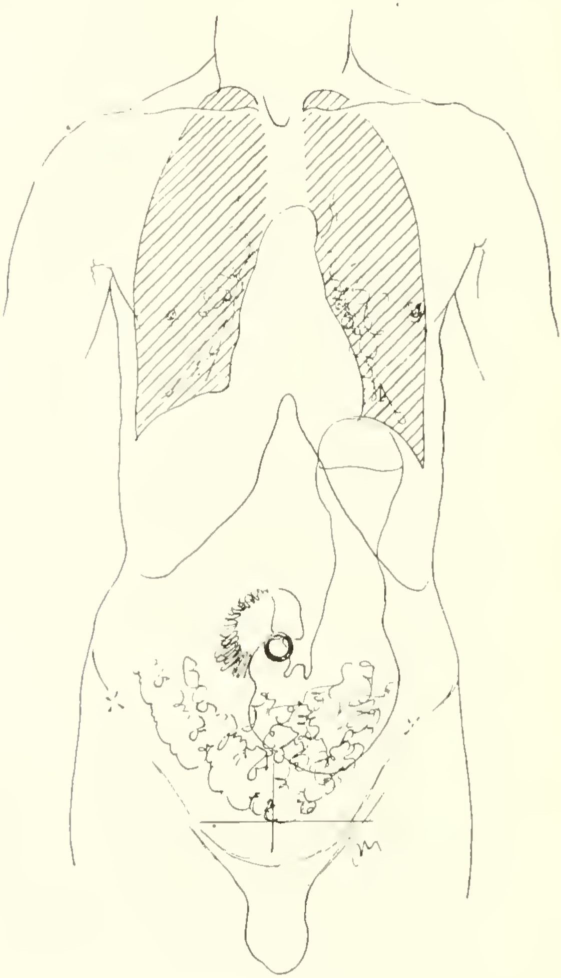

Fig. 74.—The hyposthenic to asthenic habitus, a sub-type. While essentially hyposthenic (Fig. 69) there is considerable suggestion of the asthenic (Fig. 67) in this type. This is shown in the long thorax with somewhat narrow intercostal angle but especially by the relatively wide pelvis, which, with the vertical heart and moderately low position of the somewhat atonic abdominal viscera, distinctly suggest the asthenic. (Mills.)

essentially the stomach of the hypersthenic (Fig. 66) his atonic stomach that of the asthenic (Fig. 67). The orthotonic stomach corresponds to the sthenic (Fig. 68), though less exactly than do the other stomach forms to their parallel habits. The hypotonic stomach is the stomach of the hyposthenic (Fig. 69). The classification proposed may be used as a division of all subjects into two types in which sthenic or asthenic characteristics are dominant, as a division into four major types, or may include a classification

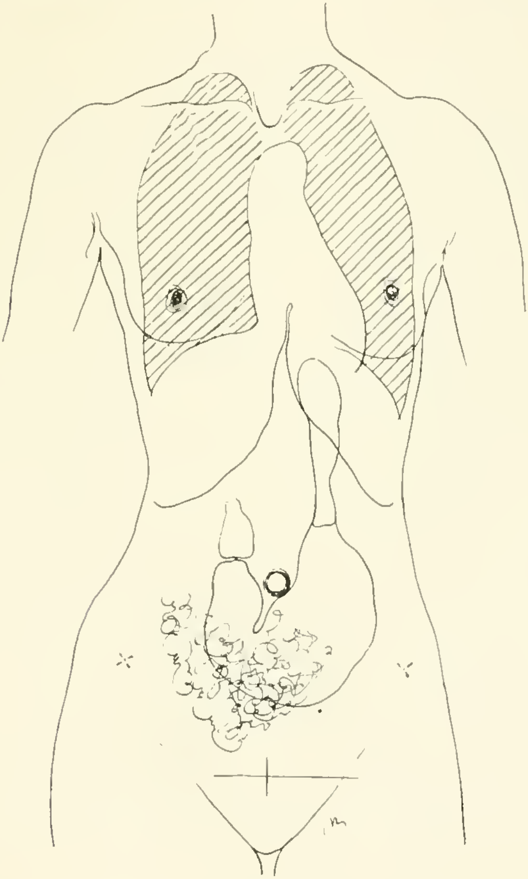

Fig. 75.—The asthenic to hyposthenic habitus, a sub-type. Essentially characteristic are those of the asthenic (Fig. 67) though there is a suggestion of the hyposthenic (Fig. 69) in the only moderately wide pelvis and the lack of that extremely low position of the stomach found in the pure asthenic. (Mills.)

of sub-types. Our classification may be illustrated by the following schema:

<table>
<tr><th colspan="6">Dominant Types</th></tr>
<tr><td>Hypersthenic</td><td colspan="2"></td><td colspan="2"></td><td>Asthenic</td></tr>
<tr><th colspan="6">Major Types</th></tr>
<tr><td>Hypersthenic</td><td colspan="2">Sthenic</td><td colspan="2">Hyposthenic</td><td>Asthenic</td></tr>
<tr><th colspan="6">Sub-Types</th></tr>
<tr><td>Hypersthenic tending to sthenic</td><td>Sthenic tending to hypersthenic</td><td>Sthenic tending to hyposthenic</td><td>Hyposthenic tending to sthenic</td><td>Hyposthenic tending to asthenic</td><td>Asthenic tending to hyposthenic</td></tr>
<tr><td colspan="4">Groups in which sthenic characteristics are dominant.</td><td colspan="2">Groups in which asthenic characteristics are dominant.</td></tr>
</table>

The sthenic type (Fig. 68) may be best described as one in which the peculiarities of that type heretofore described as hypersthenic are present but in a less marked degree—"Sthenic" subjects are heavy powerful individuals of generous body architecture, deep chests, wide intercostal angle, high digestive plant, and having more than an average degree of visceral tonus. They are a common type. Most of the heavy robust persons of one's acquaintance are sthenics. The lung fields of the sthenic resemble those of the hypersthenic quite closely in that they are also relatively wider at the base and narrower in their upper zones. Their longitudinal dimensions are short, less so than in the hypersthenic but more so than in other types. The diaphragm approaches the horizontal. The heart is less transverse than in hypersthenics but more so than in lower types. It does not appear as submerged in the diaphragmatic contour as in the pure hypersthenic. The intercostal angle is obtuse, usually about ninety degrees. The ensiform is well developed. The alimentary tract is high in position. The stomach is entirely or nearly entirely above the umbilicus, or perhaps better, above the level of the anterior iliac spines. The transverse colon is, as a rule, above the same landmarks (standing position). Other parts of the intestines occupy a proportionately high position. Visceral tonicity is of a high degree. Alimentary motility is fast.

The remaining type is the hyposthenic (Fig. 69). Subjects of this type are the most difficult of classification of any type because while the general bodily characteristics are more sthenic than asthenic, the chest and visceral arrangement in a large proportion of cases resembles that of the asthenic in the form of the lung field, general direction of heart axis and low position of the abdominal viscera. However, while thoracic and abdominal topographies resemble those of the asthenic, the visceral forms are not those peculiar to that type (Figs. 69 and 67). Those of hyposthenic type are generally somewhat frail in physique, in fact many women of this type would at first sight seem to be of asthenic habitus. They lack, however, certain peculiarities of such type especially the disproportionately wide and capacious pelvis. Each lung field is more vertical and wide in its upper zone than in those types in which sthenic characteristics are marked. The diaphragm is quite sloping. The heart approaches a vertical position, often has quite the peculiarities of the hanging heart. The intercostal angle is narrow; usually less than ninety degrees; the ensiform rudimentary or lacking. The alimentary viscera are rather low in position, a considerable part of the stomach being below the umbilicus. The intestines occupy a relative position; alimentary tonus is of but fair degree and motility of average or less than average rate.

The various types are better appreciated by the accompanying diagrams, each of which is the accurate scale record of an individual, than by description. It is better at first to accustom oneself to classifying all subjects into one of the four major types without attempting to use the sub-types.

The recognition of the different types is a matter of experience and practice and is often difficult. In some cases a subject is almost impossible of classification owing to a complexity of characteristics; the thorax may be of one type, the abdomen and visceral topography of another; such are, however, very much in the minority. A most confusing type is one in which the general habitus is one of the intermediate types, yet the degree of visceral tonus owing probably to an unusual balance in the autonomic nervous system is extreme or deficient giving the different parts of the alimentary tract the position and form of a higher or lower type.

To summarize, physical and visceral types are best recognized by an appreciation of their extremes: the hypersthenic and asthenic, all other types represent intergradations between these two. Secondly, by the recognition of two main intermediate types, the sthenic and hyposthenic, and thirdly, by variations of these four types in the form of certain sub types primarily one of the four main types but tending to the next higher or lower type in the scale.

CHAPTER XIV.

THE DIAGNOSIS OF EARLY PULMONARY TUBERCULOSIS: HISTORY AND CLINICAL SYMPTOMS.

Meaning of Early Diagnosis.—There are many misconceptions regarding the early diagnosis of tuberculosis. These have been largely unavoidable up to the present time, because our picture of this early condition has been based on a faulty conception of the disease.

What we mean by early diagnosis of tuberculosis may not be, in fact rarely is, a diagnosis made soon after the bacilli have entered the body. It may be months, often years, after the original infection has occurred that the patient observes the first symptoms and presents himself for examination. What we have been hitherto considering as early tuberculosis, considered from the standpoint of the time when infection occurs, is really late tuberculosis in most instances. It is a condition in the life history of the disease which represents not a primary invasion but an extension to new tissue; and, usually, a renewed activity in one of these extensions.

Tuberculosis is primarily a disease of the lymphatic system, the time of the infection being childhood (see page 84). No matter where or how bacilli first gain entrance to the body they pass into the lymphatic channels and are screened out by the lymphatic glands, those of the mediastinum most frequently. This constitutes the truly *incipient stage* of tuberculosis. These early mediastinal infections are found, as a rule, postmortem; but they may be inferred by a positive tuberculin test when infection of other organs has been eliminated. Infection of the glands is rarely physically demonstrable until the disease has existed for some time and the bacilli, finding themselves suited to the new soil, have already multiplied and produced a somewhat advanced pathological process. Degeneration of the interscapular subcutaneous tissue suggests it.

There is a greater similarity between tuberculosis and syphilis than is generally believed. We are told how syphilis may simulate so many other diseases in its clinical picture. Tuberculosis does the same, as I shall describe when discussing clinical symptoms. Syphilis has been divided into the primary, secondary and tertiary stage. Tuberculosis may also be so divided. Ranke, of Munich, likens the early lymphatic form of tuberculosis, the invasion, to the primary stage of syphilis; the extension to new tissue,—it may be to distant organs such as the lungs, kidneys, bowels, and meninges,—to the second stage of syphilis; and the advanced lesions with their destructive processes and general systemic disturbances to the gummatous or tertiary stage of that disease. This comparison is of more than usual interest to clinicians and at once impresses forcibly upon the medical profession facts which are of paramount importance to a rational understanding and the early detection of tuberculosis, viz.; that it is a disease of long duration, presenting a varied picture in its pathological aspects at different stages, and presenting a symptom-complex, requiring careful study; and, further, that the so-called early diagnoses are diagnoses of conditions which present an advancing process.

The problem of the diagnosis of early tuberculosis as recognized today, then, is that of detecting the disease in what might be termed the second stage of its development, the stage of increased activity as applied to the lymphatic glands or of extension to new tissue, or of any successive increased activity or extension from a previously quiescent focus.

Clinical Tuberculosis.—The writer has been in the habit of speaking of the condition just mentioned as *clinical tuberculosis* in order to differentiate it from the primary infection (the truly incipient tuberculosis) and primary metastasis in the lung, which so often becomes quiescent; both of which are often spoken of lightly as anatomical tuberculosis. It is well, however, to call attention to the fact that our best opinion today supports the idea that much of the "clinical tuberculosis" is an extension from the so-called anatomical tuberculosis, showing that the latter condition deserves far more consideration at the hands of clinicians than it now receives.

This conception puts new meaning into early diagnosis, and impresses upon clinicians the seriousness of the advanced and advancing condition which we have been wont to consider as an early manifestation. While experience shows that many of those who suffer from clinical tuberculosis will get well, even by the adoption of simple measures, the fact that one-tenth of the human race still dies of this disease is sufficient to demand that clinical tuberculosis be recognized and treated seriously.

Relationship of Clinical Diagnosis to Infection.—This shows clearly that if we would understand early diagnosis, it is necessary to study infection; to know when it takes place and what occurs when it has taken place. It is not sufficient to know that there is a pathological thickening of the tissues, accompanied by certain signs and symptoms; it is just as important to know that the cells of the body are changed and sensitized toward further attacks of the tubercle bacillus. If a subsequent inoculation of bacilli takes place, the phenomena which occur as a result of the struggle between the invading bacilli and the invaded organism are not the same as those in the primary infection. If the numbers of bacilli in the subsequent inoculation are sufficiently large, a recognizable train of symptoms occurs which is due to the struggle between the invading bacilli and the cells of the body which are now endowed with specific defensive powers and the antibodies which the cells produce for the specific purpose of warding off the invasion. If this subsequent inoculation is made experimentally by injecting bacilli into the soft tissues of an animal already tuberculous (Koch's experiment, page 82) a local ulcer forms, the bacilli are cast off, and the regional lymph glands are not even infected. If the superinfection is made through the blood or lymph stream, the bacilli settle somewhere in the tissues; and the reaction (specific cellular reaction) between the tissue cells and the bacilli again shows differently from what it does if it is a primary infection, although the difference may not be so evident as it is when the bacilli are injected into the tissues. Many of the bacilli are destroyed; and, while if the numbers are large enough, infection occurs, yet the process assumes a chronic form. In guinea pigs these subsequent infections or superinfections, as they are called experimentally, show evidence of cavity formation and other signs of chronicity while the primary inoculation develops acute disseminated tuberculosis. The nature of chronic tuberculosis with its tendency to chronicity and cavity formation is probably determined by the specific defensive properties with which the cells are endowed (sensitized condition) and must be thought of as analogous to the phenomena described by Koch in connection with a secondary infection of a previously tuberculous guinea pig. All abortive, as well as chronic forms of tuberculosis, whether of the fibroid, or fibro-ulcerative form, must be looked upon as having this specific tissue reaction as a determining factor.

The experimental work which has been done in recent years, when interpreted clinically, goes to show that the most of our instances of clinical tuberculosis are reinfections in the sense that they are infections which have taken place in bodies which have been previously infected. They are not necessarily infections by new bacilli coming from some source outside of the body of the individual, but most probably extensions from a focus within, which may have been the result of a recent infection or one which occurred months or years previously (see page 85). The probability of this is established by pathological and clinical evidence both of which go to show that the infection of the human race with tubercle bacilli is almost universal by the time the fifteenth year has been reached (see pages 78 and 100).

The phenomena which present themselves at the time of the reinfection vary and probably depend upon the degree of sensitization (specific cellular defense) present, the numbers and virulence of the invading bacilli, the suitability of the soil as a culture medium, and the character of the tissues invaded. Thus we may explain the varied nature of the symptoms and the differences in the pathological conditions which we have gradually learned to recognize as accompanying the onset of clinical tuberculosis. Thus we may explain the fact that we so often fail in recognizing the early manifestations of clinical tuberculosis.

Clinical Diagnosis.—It will be inferred from previous discussion that the diagnosis of early clinical tuberculosis takes into consideration two distinct conditions; first, the diagnosis of the disease when it first invades the lung tissues, and, second, the diagnosis of the disease when it is a renewed activity in an old focus of infection which may have been quiescent for a longer or shorter period of time. While these two conditions may present similar diagnostic data; on the other hand, they may present data, particularly that which is obtained on physical examination, of a very divergent nature. I will endeavor to make this plain as my discussion proceeds in the following chapters.

I shall take up each of the recognized methods of obtaining information and discuss it and the data obtained by it and endeavor to show how the findings vary under different conditions, hoping thus to make clear the application of the measures discussed.

There is no stereotyped way of making a diagnosis of early pulmonary tuberculosis. The diagnosis is not always based on the same conditions. It is not based on data derived from any particular procedure, but it is an opinion formed after all available data have been obtained and analyzed. He who attempts to find the same symptoms or the same physical signs in every instance before he will make a diagnosis of early pulmonary tuberculosis fails to grasp the nature of the process which he is attempting to discover. The patient suffering from early clinical tuberculosis usually shows only a few of the many symptoms which may be found accompanying this condition, and these may not be constant. They are now present for a time and then may disappear, giving the patient the idea that they are gone, as described more fully in Chapter XXI. Variability is necessarily characteristic of symptoms and signs in this disease because conditions are so varied. This can readily be appreciated from the discussion which has preceded.

The diagnosis of early clinical tuberculosis requires time for examination, skill in obtaining the proper data, and judgment in weighing the data when obtained.

Family History.—The family history was formerly of first importance, because it was thought that tuberculosis was handed down as an inheritance from one generation to another; but now that we know that this disease is an infection which occurs almost wholly after birth we attribute a different meaning to it.

The history of open tuberculosis in a family, if it has offered opportunity for intimate and prolonged association, between the one affected and the patient now under examination, is very important, and especially if this association took place during the early years of the patient's life. The reason why so much importance is attached to the intimate association with open tuberculosis in early childhood is because the closer the contact the greater the danger of infection with large numbers of bacilli; and, the greater the numbers of bacilli in the infecting inoculation, the greater the danger of the disease overcoming the natural defensive powers and assuming serious proportions. While civilized people are universally exposed to tuberculosis, those who associate intimately with it are exposed to the greatest extent. This is illustrated by the statistics of Cohn, in the following table, which show that all children in tuberculous families are infected prior to the fifteenth year, while probably 10 or 15 per cent of those in non-tuberculous families escape. Cohn, cited by Römer[1] gave the cutaneous tuberculin test to 273 children of tuberculous parents and found as follows:

Of those from	2- 3	years,	66	per	cent	gave	a	positive	reaction.
	4- 5	”	60	”	”	”	”	”	”
	6- 7	”	77½	”	”	”	”	”	”
	8- 9	”	77	”	”	”	”	”	”
	10-11	”	80½	”	”	”	”	”	”
	12-13	”	89.9	”	”	”	”	”	”
	14	”	100	”	”	”	”	”	”

The probability is that the children of tuberculous families also have a greater immunity than those of the non-tuberculous families, although a greater per cent of them die of tuberculosis. The assumption on which we base the opinion of greater immunity is the fact that if these children in tuberculous families withstand the immediate effects of the relatively larger doses of bacilli, their infections so often present abortive and chronic forms of tuberculosis. On the other hand, the greater mortality usually occurs in the acute forms of the disease such as meningitis and generalized tuberculosis which occur during the early years of life before specific cellular defense has been obtained. Von Ruck has shown that specific resistance to tubercle bacilli is transmitted by the tuberculous mother to her child.

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[1] Brauer's Beiträge zur Tuberkulose, vol. xxii, 1912.

What are we to say of the danger of intimate association as it occurs in the family of the tuberculous, particularly in adult life? Our newer studies of tuberculosis show that there is a high degree of immunity developed against the bacilli by the time adult life is reached. And we are permitted to infer that this is sufficient to protect the individual against all ordinary inoculations which might come. It is also probable that instances of tuberculosis as we find them in adult life are largely due to extensions from foci within the body rather than inoculations of bacilli from without. The real danger, however, comes from bacilli entering the tissues in sufficiently large numbers, whether from without or within, to break down the protective immunity and cause a new infective process to be set up. The greatest danger of such a thing happening comes when the individual is exposed most intimately to great numbers of bacilli, such as in the family where tuberculosis is present in the advanced destructive stage. Therefore, when the history of association with open tuberculosis in the family is positive, it is the examiner's duty to find out how long such association has existed, how intimate it was and at what period of life it occurred. A continuous intimate association with one in the advanced open stage of the disease may be of great importance at any age, but it is particularly suggestive during the early years of the patient's life.

CLINICAL HISTORY.

In order to make a diagnosis of clinical tuberculosis early, it is necessary for the examiner to disabuse his mind of the long supposed fact that a tuberculous patient must necessarily be of the phthisical habitus with flat chest, be run down, and be suffering from a low state of vitality. Such a history will be obtained in many cases, but not in all. There are many patients suffering from early clinical tuberculosis who are of robust build, who have been working hard and doing their work easily, and who show none of the usual stigmata that are assigned to those suffering from this disease. The old teaching that any man may have syphilis has its counterpart in tuberculosis. Any man may have tuberculosis; and, when it is present in its early stage at the time when it is most curable, the patient does not necessarily differ in his appearance from other members of society.

History of Past Illness.—Most patients who present for examination are suffering from renewed activity in an old focus or from an extension of the disease to new areas. Therefore, it is important to inquire carefully whether the patient at any time during his previous years suffered from similar symptoms. It is not uncommon for the patient, after a little reflection, to recall previous attacks which were similar; or, to recall other symptoms or attacks which were most probably associated with previous periods of activity in the same or other tuberculous foci. A previous pleurisy, or tuberculous bronchitis, or spitting of blood or fistula is not uncommonly found in the previous history. antedating the present illness from one to twenty years.

These attacks are usually characterized by other names, sometimes through ignorance and in other instances with intention to deceive. ''La grippe,'' ''bronchitis,'' ''neurasthenia,'' ''anemia,'' ''malaria,'' ''a run-down condition,'' ''intercostal neuralgia,'' ''bleeding from the throat,'' are terms which are frequently employed in comforting and lulling the patient, who is suffering from early clinical tuberculosis into false security. We can hardly conceive of anyone having constantly repeated attacks of *la grippe; bronchitis* which hangs on and comes frequently is always suspicious; *neurasthenia* demands a diagnosis and has tuberculosis for its etiological factor oftener than is generally believed; *malaria* should not be confidently assigned as the cause of the general lack of ambition which is found in malarial districts, for these patients also are subject to tuberculosis and other diseases which cause the same symptoms; *a run-down condition* is suggestive of many things but demands that the physician search until the true cause is found, and if repeated or at all persistent should call for careful search for tuberculosis; *intercostal neuralgia* is frequently diagnosed when pleurisy of tuberculous origin is the real condition present; and *bleeding from* *the throat* is the sedative which throws many people off their guard and allows an early tuberculosis to creep on to an advanced hopeless condition. The history of any of these conditions during the past life of a patient is extremely suggestive of previous attacks of active tuberculosis.

Slow recovery from other diseases should always excite suspicion. This is particularly pertinent since our recent epidemic of influenza. Some of the common symptoms will often appear if tuberculosis is present and careful physical examination of the chest should be made. The tuberculin test cannot always be relied on under the circumstances. If positive it is valuable; but if negative, it should be accepted with great reservation (see page 504).

Sometimes a definite history of a tuberculous lesion elsewhere in the body, either at the present time or sometime in the past can be obtained. If so, it should not bias the examiner, but cause him to be careful in weighing it along with other evidences.

Present Illness.—Hawes[2] rightly emphasizes the importance of asking the patient how long it is since he was perfectly well. The writer has found this of great importance. The patient does not know when he became ill of the tuberculous process because the symptoms at first are so slight that he does not recognize them as making him ill until the early stage has passed. In answer to this question, however, he will often go back weeks or months, and sometimes even years before he finds the time when he really felt well. Some patients will be unable to give any history of previous disease, the active symptoms coming on without warning. This sudden explosive type of tuberculosis is usually overlooked; coming as it does without the usual antecedent history of being run down, and without stomach and nervous manifestations, tuberculosis is not thought of. From the marked rise of temperature present it is often called typhoid, or, if the lung is examined, it is diagnosed as pneumonia or la grippe.

Classification of Early Symptoms.—The symptoms belonging to tuberculosis are varied and appear as expressions of disturbance in many different structures and organs. When analyzed, however, all of the twenty or more symptoms can be classified according to their etiology in three groups as follows:

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[2] Early Pulmonary Tuberculosis, William Wood & Company, New York, 1913.

GROUP I	GROUP II	GROUP III
SYMPTOMS DUE TO TOXEMIA.	SYMPTOMS DUE TO REFLEX CAUSE.	SYMPTOMS DUE TO TUBERCULOUS PROCESS *per se.*
Malaise	Hoarseness	Frequent and protracted colds
Run down condition	Tickling in larynx	Spitting of blood
Lack of endurance	Cough	Pleurisy
Loss of strength	Digestive disturbances (hypermotility and hypersecretion)	Sputum
Nerve instability	Loss of weight	
Digestive disturbances (hypomotility and hyposecretion)	Circulatory disturbances	
Loss of weight	Chest and shoulder pains	
Increased pulse rate	Flushing of face	
Night sweats	Apparent anemia	
Temperature		
Blood changes		

The above classification was first published by the author in January, 1914.[3] Since that time I have made slight changes in the arrangement of the symptoms. By so grouping the symptoms we gain a fuller comprehension of the disease and grasp the situation which presents with greater understanding.

General Characteristics of the Toxic Group.—It will be noticed at once that this group of symptoms is expressive of nerve and endocrine stimulation; that there is general inhibition in the enteral system; that the symptoms are expressed widely through the body; and that they form the symptom-complex expressive of general nerve and endocrine unbalance and peripheral sympathetic action. It will also be noted that this group of symptoms is not alone characteristic of tuberculous toxemia. It can as well be an expression of an infection in a tonsil, the prostate, the fallopian tube, or an intestinal toxemia, or one of the acute toxemias, as of tuberculosis; consequently, when taken alone is of limited value as diagnostic evidence of the presence of active tuberculosis.

The future will reveal more fully the relationship between the toxic state and the secretion from the ductless glands. There is considerable disturbance in glandular functions. The adrenal glands are supplied by branches from the splanchnic group of the sympathetics, and respond with increased secretion when the sympathetics are stimulated. Not only do they respond to sympathetic stimulation, but the adrenin when circulating in the blood stream produces action of the same character and in the same organs as that produced by the sympathetics. The enlargement of the thyroid, as frequently observed during early clinical tuberculosis, suggests that this gland is probably called upon for extra service in the presence of infection. This is also supported by its enlargement in the presence of other infections.

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[3] Northwest Medicine, Jan., 1914.

No matter how the symptoms are produced or prolonged, the syndrome of toxemia is that of central nerve stimulation plus general discharge through the sympathetic nervous system and an endocrine unbalance.

As the degree of toxemia varies, so there is a varying action shown in the vasomotors which at times shows itself as a vasoconstriction of the cutaneous vessels interfering with the elimination of heat. There is also a general sympathetic stimulation throughout the gastrointestinal tract which results in inhibition of motility and secretory activity. While such action is common when toxemia is present, it is often prolonged by anxiety, fear, worry, and pain. Tuberculosis goes through periods of acute activity now and then, which are followed by periods of quiescence. In the early stage, and up to the time that softening begins, clinical tuberculosis is in a state of quiescence in by far the greater portion of the time. And even then, unless the area of softening be a large one, the periods of quiescence are relatively long.

The symptoms belonging to the toxic group should pass away when the acuteness of the process is over; or when the body has established an ability to properly counteract these toxic influences. When either of these states has been reached, however, toxemia may still be able to produce its symptom-complex by faulty methods of living on the part of the patient, particularly overexertion with its attendant increased heat production and autoinoculation. So may the same group of symptoms be prolonged by such depressive states as pain, anxiety, fear, disappointment, and discouragement.

So, it will be seen, that, while this group of symptoms is of great importance in the diagnosis of early active tuberculosis; yet its presence alone is not sufficient to make a diagnosis. It must be accompanied by other symptoms and other signs. It is equally, if not more, important to remark the opposite of this—*that active tuberculosis may be present without the presence of the symptoms due to toxemia.*

It is of greatest importance to early diagnosis that this fact be known. To that end let us recall our pathology. Early pulmonary tuberculosis is an infiltration in the lung tissue. The bacilli are embedded in the tissues and are surrounded by new cells. The process becomes acute only when the bacilli multiply and toxins diffuse into the adjacent tissues. If this process is carried sufficiently far, necrosis takes place. This necrosis does not involve all of the infiltrated tissue, but only a circumscribed portion. When caseation occurs, as it so often does, or when the process has subsided without caseation taking place, the acute symptoms of toxemia disappear; yet the same pathological changes may soon take place in other areas, so we are not justified in looking upon the process in any other manner than as an active one. That this characterization is correct is usually shown by other periods of acute pathological changes with attending toxic symptoms taking place after intervals varying in length from a few days to weeks or months. It is also proved by the fact that the symptoms due to reflex cause, likewise the physical signs due to the same cause, continue to manifest themselves over periods of months after the toxic group of symptoms has disappeared. This is well illustrated in the cases described in The Tuberculosis Clinic, Chapter L, Vol. II.

SYMPTOMS DUE TO TUBERCLE TOXINS.

There is no regularity in the severity of the symptoms based on toxemia because the amount of toxins differs so markedly in different patients and under different conditions. While this can be more readily seen in advanced cases where we have the two active forms, acute caseous tuberculosis and the slow fibro-ulcerative form, it is also evident in the early cases. One patient will suffer greatly and show most of the symptoms mentioned in Group I, and another will hardly realize that he is ill and scarcely complain at all. The latter patient should have the best chance of recovery, for the inhibition of his conservative forces, as produced through stimulation of the sympathetic nervous system, is less marked, or, as we would naturally say, his disease is less severe. This advantage, however, is too often sacrificed by the delayed diagnosis and unwarranted certainty on the part of the patient, either that he is not ill or that his illness is so slight that he will recover without following a careful routine.

Malaise, Nervous Instability, a Feeling of Being Run-down, and Lack of Endurance.—These are common symptoms of active tuberculosis. They are particularly expressive of the effect of the toxins upon the nervous and endocrine systems, although they may be caused by all the forces which tend to lower the patient's vitality. When an old tuberculous process becomes active, or a new infection occurs, patients are very apt to note that they tire more easily than formerly. They suffer from a languor which seems unexplainable. This, at times, almost amounts to an aching. Patients sometimes feel that they do not want to move or be disturbed, and yet they can see no reason for it. Sleep and rest do not refresh them. They seem to have an indefinite feeling due to what is to them an inexplainable cause. It is nothing they can explain, nothing that they can fully grasp; yet there is a consciousness that something is the matter. Sometimes they are taken to be lazy by their friends, and even believe it themselves. When the toxemia is very severe these symptoms are increased. Speaking of languor, Minor well puts it: "The whole body seems filled with tiredness." They note that their disposition is changing. They are irritable and more easily disturbed than usual. They often lose ambition and assume a "don't care" attitude. It is such an effort to do things that they find themselves neglecting duties which they have always assumed cheerfully.

Tasks which were formerly easy are now accomplished with difficulty. A little effort is followed by exhaustion from which recovery is slow. The patient then finds himself in a run-down condition, a state of low vitality, from which he seems unable to pull himself together. At first the patient does not feel like doing; later he cannot do. His endurance is gone. This state is often diagnosed as *neurasthenia*. To be sure, neurasthenia is present. But this is not a diagnosis. Neurasthenia has a cause. It is a condition, not a disease. Tuberculosis is very often the cause and should always be thought of, especially if any other signs, such as rise of temperature, and loss of weight, and particularly symptoms belonging to the other groups, are present.

Gastrointestinal Symptoms.—The toxic symptoms on the part of the gastrointestinal tract in early clinical tuberculosis manifest themselves as a general inhibition of action in all the functions of the alimentary canal. They are not always constant, nor are they always present in the same degree of severity, yet an active process is often accompanied by some degree of inhibition of the functions of the digestive tube. The patient may note a *capricious appetite;* or the appetite may be absent. At other times he may have little or no noticeable disturbance. During the periods when the toxemia is most acute, however, there is usually at least a slightly coated tongue, a deficiency of gastric and intestinal juices and a deficiency of gastric and intestinal motility, all of which is an expression of general sympathetic disturbance. *Loss of weight* usually follows this digestive disturbance although it is often difficult to find accurate data on this point as people do not weigh regularly, often not for years. In early tuberculosis this loss only amounts to a few pounds. When there is a loss of fifteen or twenty pounds of weight as a result of the tuberculous process, it indicates that the disease is not early. Such a loss in weight must have a severe pulmonary involvement back of it. The infection is either very virulent or widespread, or both; or the inhibited function may be due to depression.

It is particularly important for those who are attempting to diagnose early tuberculosis to bear in mind that both men and women attain a maximum weight for their early years when about eighteen or twenty years of age. After holding this a year or two they will fall back five, ten, and sometimes fifteen pounds and then hold this as their normal weight until after the third decade has been reached. It is very important to bear this in mind when taking histories for it would be manifestly erroneous to consider this maximum as the normal weight, or this loss as pathological. Other causes for loss of weight must always be considered. This symptom has no value unless accompanied by others, and at about the age of twenty it may be due to the natural decrease in weight which I have just mentioned.

Night Sweats.—In our attempt to offer a physiological explanation of the sweating which occurs in tuberculosis, we are confronted with the fact that there is still an uncertainty in the minds of physiologists as to the manner in which sweating is produced. There are certain facts which suggest that sweating is at times due to stimulation of the sympathetics, and, again, to stimulation of the parasympathetics. It accompanies toxemia, fear, rage, and other symptoms which we know are definitely due to sympathetic stimulation. On the other hand, sweating is found as a symptom of vagotonia. It is also relieved by atropin, which is the physiological antagonist of the parasympathetics.

The muscles of the sweat glands receive their innervation from the sympathetics, and when stimulated to contraction, are able to force the sweat from the glands, providing it is already produced; but we do not believe that this is the physiological explanation of sweating. There is unquestionably a definite innervation of the glands themselves; but whether this comes from the sympathetic or parasympathetic divisions of the vegetative system, or both (the latter seems certain from clinical observation), will have to be determined by future study. In tuberculosis, sweating is an accompaniment of the state of toxemia.

Rise in Temperature.—Rise in temperature can either be due to bacillary toxins or to the absorption of other protein from the inflammatory process in the lung itself. It is unquestionably due partly to some increase in metabolic activity resulting in increased heat production, but also to an interference with the elimination of heat when formed.

The discussion of the question of temperature seems more appropriate in connection with the study of advanced tuberculosis; yet, in order to make the etiological basis of this symptom clear it should be discussed at this time.

That a rise in temperature may be due to either an increased heat production or a decreased heat elimination, or both, seems evident. To explain the rationale of its production, however, is not so simple. For years my study has led me to look upon the rise of temperature which occurs in infections in general, as a conservative process.[4] It is an index to the reacting powers of the patient. This may be seen in a comparison of the behavior of the child and the old man toward the same infection, as in pneumonia. The child has a high fever and recovers, the old man exhibits a temperature of 100° to the same infection and succumbs. Crile[5] considers temperature as a part of the protective mechanism against infection. He says: "As to the mechanism which produces fever we postulate that it is the same mechanism as that which produces muscular activity. Muscular activity is produced by the conversion of latent energy into motion, and fever is produced largely in the muscles by the conversion of latent energy into heat."

I will not enter into a free discussion of the cause of fever at this time, having done so more fully in Chapter XXX, Volume II. It is necessary, however, if possible, to make plain the cause of fever, that we may be able to understand the value of this symptom in the early diagnosis of tuberculosis. During the time when tubercle bacilli are multiplying they form toxins which find their way into the blood stream. The inflammation in the lung also causes a certain chemical activity resulting in the absorption of toxic products. The destruction of these toxins is produced by chemical action which results in an increased production of heat. These toxic products also act upon the central nerve cells. They exert a particular action upon the vasomotor center and produce a general stimulation of the entire sympathetic system. As a result, we find vasoconstriction as a part of the syndrome of toxemia. As a result of the extra heat production, and from this vasoconstriction which is produced by toxemia, there results increased heat production and decreased heat elimination, and a consequent rise in temperature.

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[4] Tuberculin in Diagnosis and Treatment, C. V. Mosby Co., St. Louis, 1913, Chapter X.
[5] The Origin and Nature of the Emotions, W. B. Saunders Co., Philadelphia, 1915.

This increase in body heat tends to prevent the multiplication of bacteria which menace the organism and at the same time favors the production of specific defensive substances. Crile postulates that the effect of pain, emotions such as fear and anger, and infections, is all one—that of bringing into activity the latent energy of the body and converting it into active defense. The process is the same whether it expresses itself in a motion to escape, or in an attack upon an antagonist, or in the destruction of dangerous toxins by oxidization with an accompanying rise in the body temperature which inhibits the growth of the invading bacteria from which the toxins arise. This action takes place through the brain, adrenals, liver, thyroid, and muscles, the organs of the body which are particularly concerned in the transformation of latent into active energy.

A carefully constructed temperature curve is of great value as an aid to the diagnosis of early clinical tuberculosis during the state of toxemia, but of less value after the toxic state has passed. This cannot be emphasized too strongly. *If other suspicious symptoms are present and a carefully constructed temperature curve is characteristic, and a tuberculin test should prove positive, the reaction reaching its maximum early, the diagnosis of probable active clinical tuberculosis should be made even in the absence of physical signs on percussion and auscultation.*

In order to construct a temperature chart of any diagnostic value, care and exactness must be exercised. A carefully constructed 30 day chart will greatly facilitate the study of temperature. It is very difficult to secure an adequate picture when the temperatures are taken and placed in columns on a sheet of paper, but when a graphic 30 day chart is made, the picture is grasped at once. The chart should be made of four daily records, and should include both the maximum and minimum for the given patient for the day. It must be recalled that different patients will reach their minimum and maximum temperatures at different times of day. In one the maximum may be ten or eleven o'clock in the morning, though this is rare. Such is most commonly met in those who begin their daily activities early. It might be at two

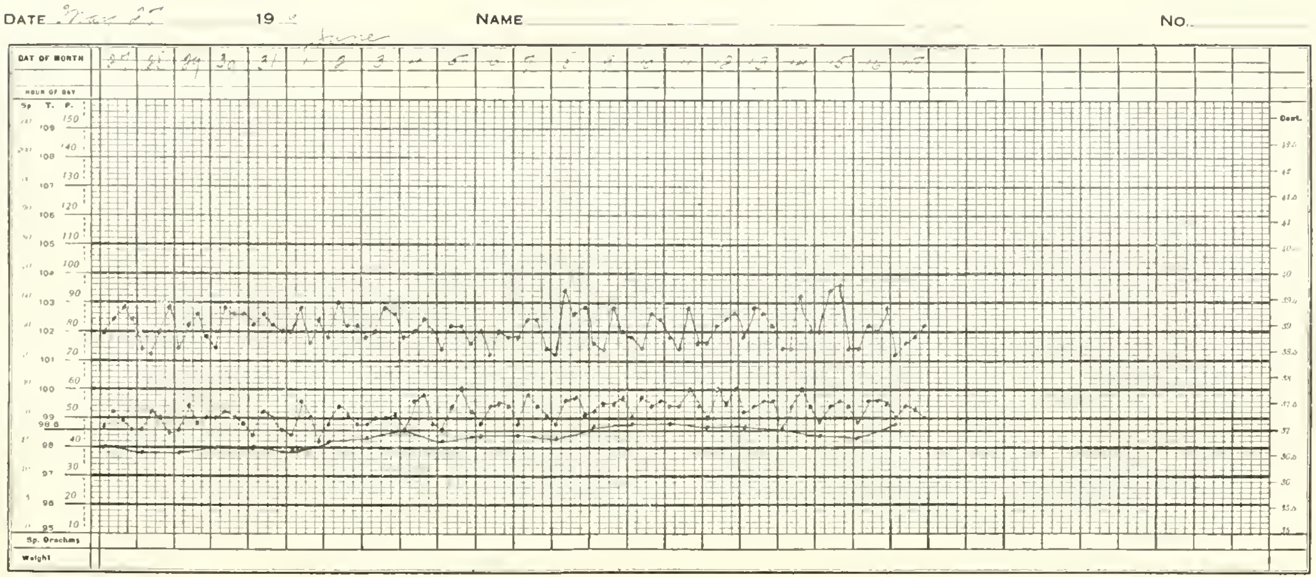

Fig. 76.—Illustrating the fact that many temperatures which show diminished diurnal variation and a high minimum record when taken at 7:00 or 8:00 in the morning, are due to the temperature starting to rise at an earlier hour than it does in the average patient. In the construction of the regular curve the patient took her temperature at 7:00 A.M., and it was always found to be about normal or even above, but when taken at 5:00 A.M., as indicated by the solid line below the regular daily minimum, it shows that the temperature at this time was one-half to three-quarters of a degree lower.

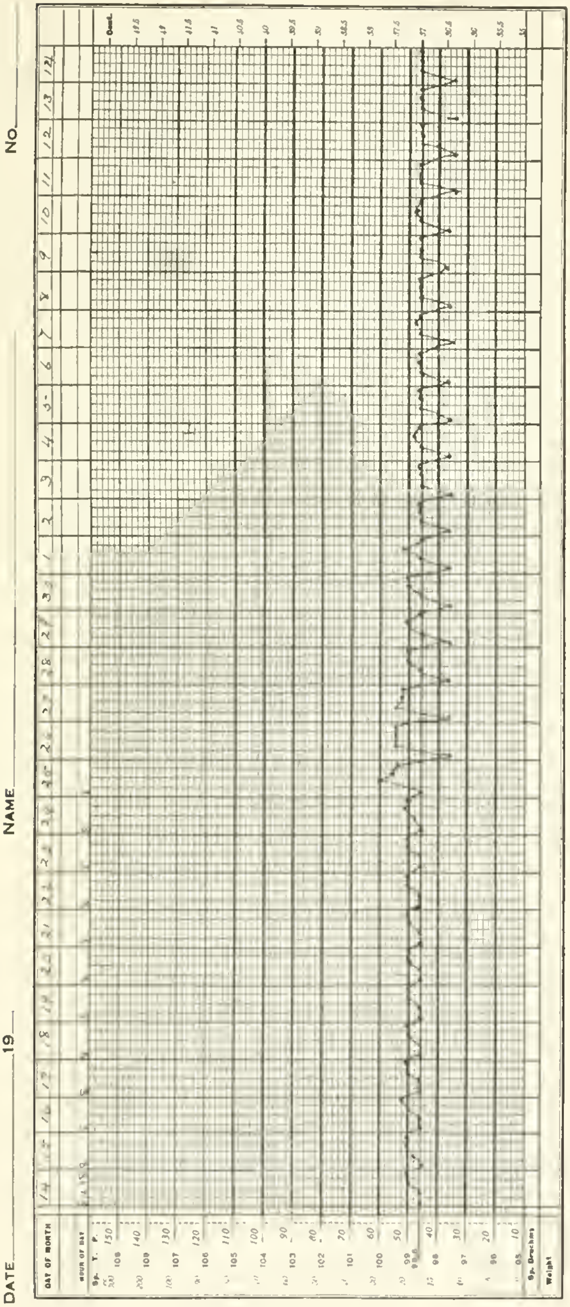

Fig. 77.—Illustrating the importance and necessity of knowing the early morning temperature in order properly to understand the temperature curve. During the first ten days shown on the chart the patient made the *first* record at 8:00 A.M. Seeing that the diurnal variation was so small, it was taken on arising as shown in the chart from the 26th on. What seemed to be an abnormally small diurnal variation proved to be a normal one when the curve was properly constructed.

in another or even at ten or eleven o'clock at night. My plan, where there is doubt, is to have a two-hourly chart made for a few days until I can see at what time the maximum temperature appears. I then choose this as one of the hours of the day when temperatures are to be recorded.

I wish also to emphasize the importance of the early morning temperature. If one will observe the temperature in the morning when the patient awakens before the activities of the day begin he will have a very important starting point for observation. The temperature at this time of day, depending on the time that it is taken, should be from 97.2° to 98° F. If we find the temperature 98.6° at this time, must be considered as above normal; but may be evidence of an early rise of normal temperature for the individual patient as shown in the Figs. 76 and 77. The normal daily variation in temperature is about one and a half degrees. Unless the patient's temperature is at this low point in the morning, he is suffering from a constant rise. It is not sufficient to take the maximum temperature for the full study of the fever curve, but the early morning temperature must be equally considered for the diurnal variation is of value in determining the stability of heat regulation. I usually construct my temperature curve from that taken on awakening in the morning, twelve, four and eight o'clock unless the maximum should fall on some other hour. Fig. 113, Vol. II, shows a normal temperature curve for the day, while Figs. 76 and 77 show the importance of the early morning record.

There are many sources of error in taking temperature. In the first place, the thermometer should be held in the mouth sufficiently long to fully register. I am in the habit of requiring patients to hold the thermometer a minimum of five minutes, no matter whether it be a one minute, two minute, or three minute thermometer. If the weather is cold and the cheeks are chilled, the temperature will not record in this time. The thermometer must sometimes be held ten or fifteen minutes to secure a full reading. It takes ten to twelve minutes to warm the mouth if one has been riding in the cold or if one has been talking in the cold air. A single observation made at the time of the visit of

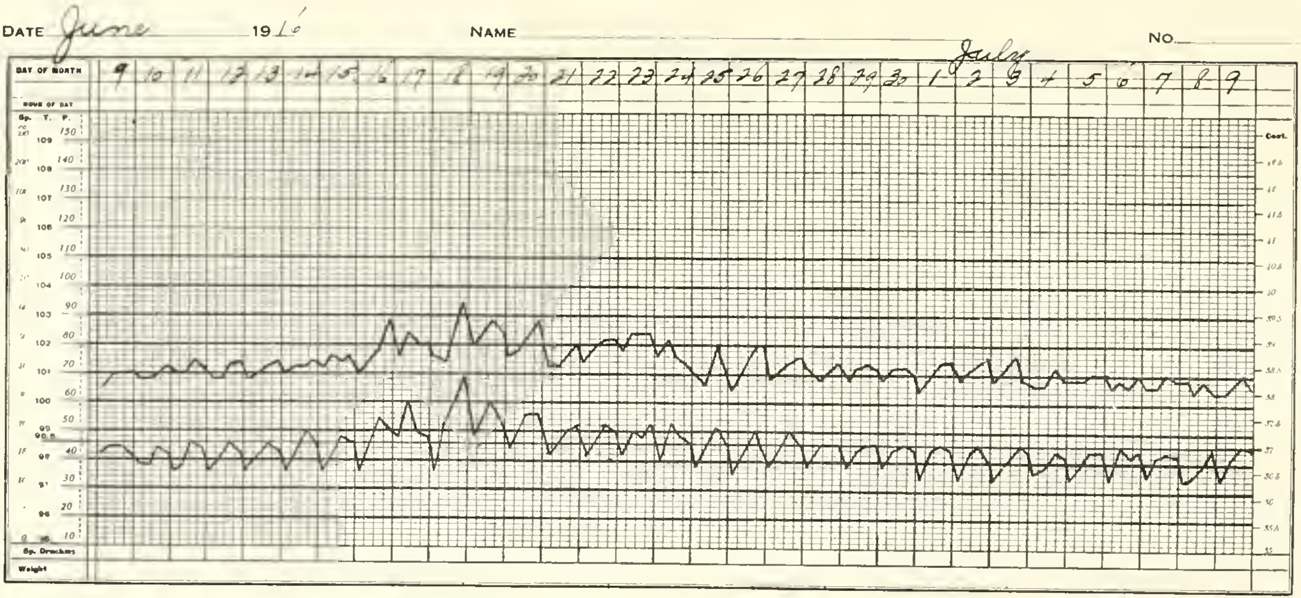

Fig. 78.—Illustrating the impossibility of judging whether a patient is suffering from clinically active tuberculosis by one day's or a few days' temperature record. This patient would have shown no elevation in temperature during the first five days and during the last twelve days of the chart. On the other days he would have shown a rise above normal.

the patient to the physician's office is of no value in determining the presence or absence of a rise in temperature.

The patient should be instructed to hold his mouth closed during the entire time that the thermometer is registering. Mouth-breathers have great difficulty in registering their temperatures. In fact, mouth temperature in a mouth-breather has little value. The rectal temperature should be relied upon under these circumstances.

There are certain peculiar characteristics of the temperature curve that should be considered.

First: Patients suffering from active tuberculosis do not necessarily have symptoms of toxemia with the maximum temperature above normal every day over a prolonged period of time. This toxemia is usually inconstant and shows waves of increase and decrease. The patient may have a rise of temperature above normal for a period of a week or more, succeeded by a like period of freedom from rise. It can be seen that if the temperature was taken during the period of acute exacerbation the patient would be said to have an elevation of temperature. However, if it were taken during the time when the acuteness of the toxemia had passed, he would be said to have no rise of temperature; consequently it is impossible to give a definite opinion of the temperature of the patient in whom early tuberculosis is suspected unless the chart be taken for a period of two or three weeks. This point is well illustrated by the chart in Fig. 78. The only time that this patient showed a rise in temperature was when he was suffering from pleurisy, the lesion being a subpleural one. (See page 377.)

Another important variation to be noted is the premenstrual rise. This elevation of temperature varies greatly. In some instances it will be extremely regular, the temperature running from 98.6° to 99° in the afternoon for the two weeks prior to menstruation and from 97.4 or 6° to normal for the two weeks following menstruation. In others the elevation of temperature will appear only a week or a few days before the menstrual time. In still others it will appear during the period and continue for a short time; but this is the exception. The cause of this most common rise in the temperature curve prior to menstruation

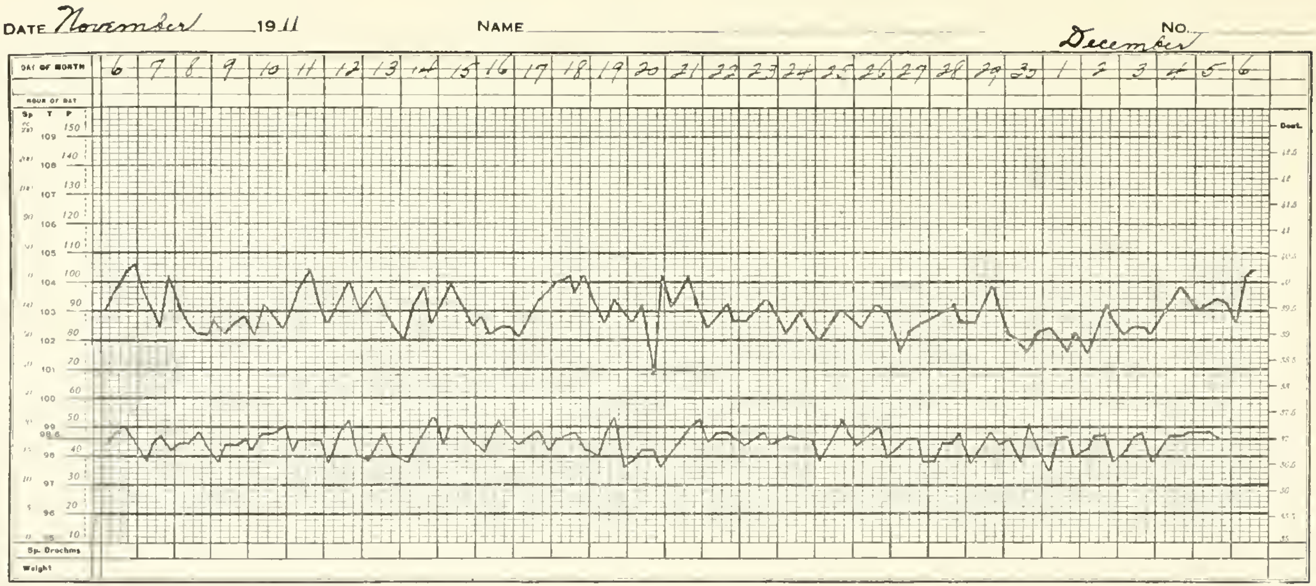

Fig. 79.—Illustrating the temperature and pulse curve of a chronic neurasthenic patient suffering from a moderately extensive fibroid tuberculosis, which at the time indicated by the chart was wholly quiescent. The irregularity in pulse and temperature may be accounted for by the nervous state of the patient. It can be seen that the height of the temperature curve is some greater from the 14th to the 27th of the month. This is the premenstrual period, but the chart as a whole is marked by many departures from the normal curve.

where the daily maximum amounts to little more than one-half of one degree Fahrenheit, we must look for in a disturbed equilibrium between heat production and heat elimination. This we have in an increased metabolism and the stimulation of the vasoconstrictors caused by the ovarian secretion produced during this time, as discussed more fully on page 195.

More marked rises in temperature which occur immediately preceding or during the period, and which continue through menstruation, may be associated with inflammatory conditions in the genital organs. This premenstrual curve, as far as I have been able to determine, appears the same in normal women as it does in the tuberculous, although I have had only a few persons who were able to keep a chart for me sufficiently long to be of value; yet this is what should be expected on physiological grounds.

If there should be a rise in the temperature curve during this premenstrual time, I would not give it, of itself, any diagnostic worth, but would wait and take the temperature again following the cessation of menstruation. The premenstrual rise is shown in Fig. 76, in which it will be seen that the temperature as a whole was about one-half degree higher from the 4th to the 17th of the month. It showed a slight decline on the 17th, the 18th being the first day of menstruation. (See page 374.)

Patients can readily be taught to take their own temperature and pulse and this is far better than to rely on the irregular observations made by the physician in his office. From one-half to one degree should be allowed for nervousness on the first visit to the physician.

A very annoying temperature curve is often shown by those of nervous temperament. It is characterized by irregularity, being up one day and down the next. While this character of curve is often met with in the tuberculous patient, the irregularities are not primarily due to the tuberculous process. Fig. 79 illustrates this rise of temperature. Such rises of temperature may continue in nervous individuals for months after the disease is arrested. It is often difficult to convince them that these rises are not serious and that they are not caused by active processes. The probable explanation is that, through the action on the nervous system, stimulation of the vasoconstrictors is produced which interferes with the elimination of heat.

There is a tendency on the part of some observers to go too far and to try to account for nearly all persistent rises of temperature as being of tuberculous origin; but when other suspicious symptoms are absent, we must remember that infections of such organs as the tonsils, teeth, appendix, and genital organs, particularly the prostate and fallopian tubes, or certain nervous conditions, can cause an elevation of temperature. Fig. 80, *A* and *B,* shows a curve in a patient who was suffering from chronic inactive tuberculosis and who developed a tonsillitis. A local infection remained in the tonsil, causing the temperature curve here shown. (See pages 382 and 383.)

Acceleration of the Pulse.—The pulse acceleration noted particularly on exertion during early active tuberculosis is a symptom which results chiefly from the disturbance of the inspiratory act and from the effect of toxins upon the sympathetic nervous system. The degree of acceleration of the pulse in early tuberculosis varies considerably according to the individual. In young persons it is apt to be greatest, but varies considerably according to whether the patient is vagotonic or sympathicotonic, according to the ideas of Eppinger and Hess. After the symptoms of activity have disappeared, the pulse returns to a normal rate, or the degree of acceleration is lessened. The peculiar characteristics of the pulse in early tuberculosis will be discussed more fully later on in this chapter in discussing the effect of reflex action; but, it should be said at this point that the degree of rapidity of the pulse is the resultant largely of sympathetic stimulation caused by toxemia and increased output of adrenin on the one hand, and reflex stimulation of the vagus produced by the inflammation in the lung, on the other hand. During periods of toxemia, sympathetic stimulation usually predominates and the pulse becomes accelerated. After the activity has passed by, either vagus or sympathetic stimulation may be most marked according to the individual. If one should expect to follow the accepted teaching that acceleration of the pulse is the particular characteristic of active tuberculosis, he could go wrong in the majority of instances when the patient is resting, because acceleration is not regularly present unless the patient is exerting or nervous.

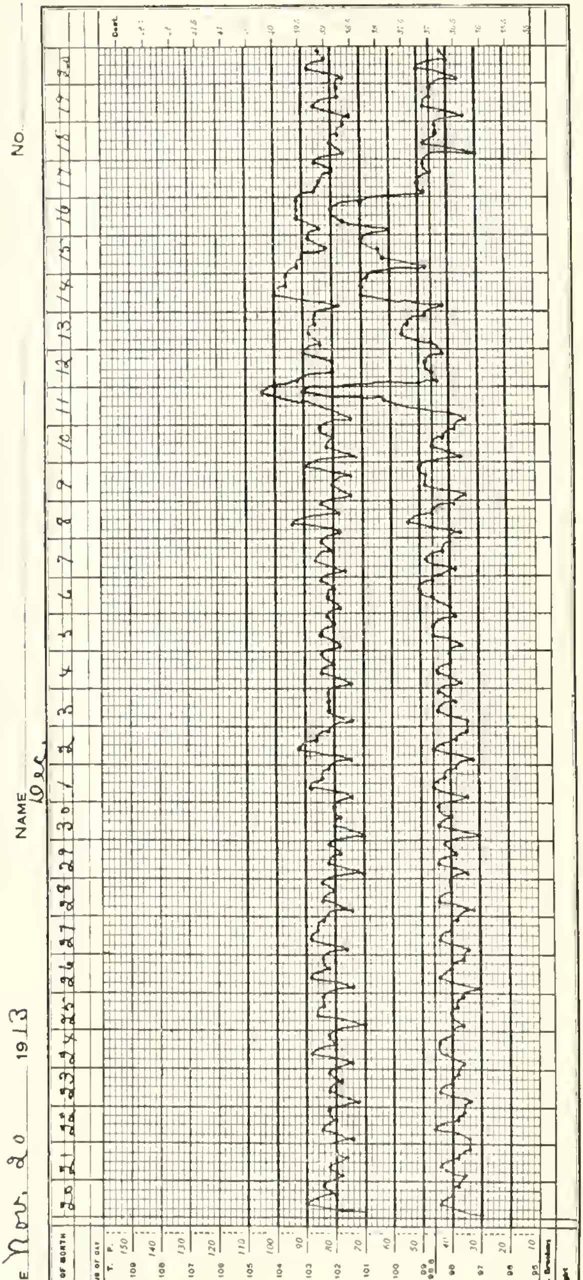

Fig. 80*A*.—Showing the influence of a complication in keeping up a temperature curve. The patient had been suffering from chronic quiescent tuberculosis. He developed a tonsillitis which was at its maximum on the 14th, 15th and 16th of December. A focal infection remained in the tonsil, which produced the rise in temperature shown in Fig. 80*B*. *A*—Curve of chronic quiescent tuberculosis with complicating tonsillitis, December 11 to 16.

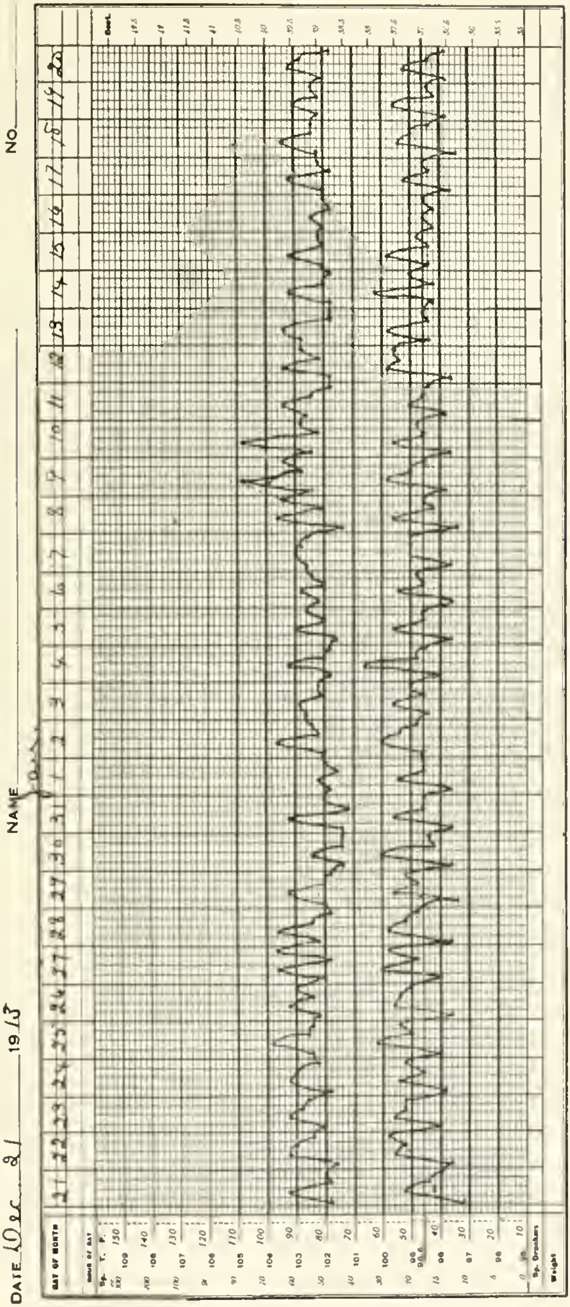

Fig. 80B.—Showing the rise in temperature as a result of focal infection in tonsil following acute tonsillitis.

SYMPTOMS OF REFLEX ORIGIN.

Symptoms of reflex origin are a result of peripheral stimulation of the pulmonary ends of both the sympathetics and vagus. That of the former results in reflex action in certain muscles of respiration and in the tissues of neck and shoulders: that of the latter in functional disturbances in other organs.

Another peculiar characteristic of the symptoms due to reflex action is that they all point away from the lung. Hoarseness, tickling in the larynx and cough point to the larynx as the cause of the trouble. The instability of the heart action sometimes points to the heart. The gastrointestinal disturbances call attention to the stomach and intestinal tract, while shoulder pains are very apt to be considered as rheumatic or neuralgic in origin. Thus, it can be seen that tuberculosis masquerades as an affection of each one of the great systems of the body; sometimes symptoms on the part of the nervous system predominating, sometimes those on the part of the circulatory system, and sometimes those on the part of the gastrointestinal tract; and still again, the symptoms may point to the upper air passages.

In my discussion of the vegetative nervous system I have stated that there is some disagreement in the minds of physiologists upon the point of reflex sympathetic action. Physiologists almost universally maintain that there can be no reflex action resulting from afferent impulses in the sympathetics without mediation taking place in the cord. There are certain sympathetic reflexes, however, which appear when the lung is inflamed, such as dilatation of the pupil, in which both afferent and efferent impulses seem to course over sympathetic neurons; but the probabilities are that mediation takes place in Budge's center in the cord.[46] In parasympathetic reflexes the sensory fibers of the vagus mediate in the medulla with efferent neurons of the vagus going to all structures supplied by it, causing reflex functional disturbances in the pharynx, larynx, gastrointestinal tract, heart or in the lung itself.

In order to understand the symptoms of reflex origin we must bear in mind that wherever the sympathetic and parasympathetic nerves meet, their action is antagonistic; consequently, we have as a result of central and peripheral stimulation of the sympathetics by toxins and adrenin an action which is antagonistic to that which results from reflex stimulation of the vagus by the inflammation in the lung. So do we find that other conditions which cause sympathetic stimulation, such as fright, worry, pain and discouragement or the condition of sympathicotonia have a tendency to counteract the action of the parasympathetics.

We must conceive, in studying the symptomatology of tuberculosis, of there being constant forces at work to disturb the balance between parasympathetics and sympathetics, which results now in increased parasympathetic, and, again, in increased sympathetic, tonus. In this way we can understand the variability of the symptoms which are present throughout this disease. Not only is this variability present in tuberculosis, but it is present in all inflammatory diseases of internal viscera. The same nerve influences can be worked out for each disease.

I would also call attention to the fact that in certain individuals parasympathetic tone naturally predominates over sympathetic. In such cases parasympathetic symptoms naturally predominate. In others sympathetic tonus predominates over parasympathetic. Marked parasympathetic, the same as marked sympathetic tonus, is a sign of an unstable nervous mechanism and denotes lowered resistance. Conditions of vagotonia and sympathicotonia may either be underlying predominant factors in the patient prior to his illness or they may be precipitated as a result of the disease.

The symptoms which are of reflex origin are not constant. For example, we may have hoarseness at one time and not at another time. We may have a rapid heart at one time and a slow heart at another. There might be a good appetite with good digestion at one time, with poor appetite and slow digestion at another, depending on which division of the nervous system predominates.

Hoarseness.—Hoarseness is often found as one of the early symptoms of tuberculosis. If careful inquiry is made and careful notice is taken of the patient's voice, this symptom will be found to be present in early tuberculosis much more commonly than is generally believed. It is extremely common in advanced tuberculosis, particularly during periods of acute activity and cavity formation. Complete aphonia lasting for a few days is not at all uncommon at this time, although a marked degree of hoarseness is more apt to be present. In early cases hoarseness is slight and often overlooked. The afferent impulse which produces hoarseness travels centralwards through the pulmonary branches of the vagus. The efferent returns through either the superior or recurrent laryngeal branch of the vagus. Where the efferent comes through the superior laryngeal it shows as a relaxed condition of the cord, an inability to approximate particularly in the center, because the interference is in the cricothyroid muscle which increases the tension of the cords. Where the efferent impulse is through the recurrent laryngeal, on the other hand, we have an apparent adductor paralysis, which shows as an interference in adduction. The cord involved hangs back and fails to approach its fellow in the center. Both of these types of disturbance in the cords may be found now and then throughout the course of the disease. They are illustrated in Figs 81 and 82.

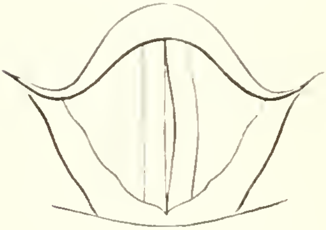

Fig. 81.

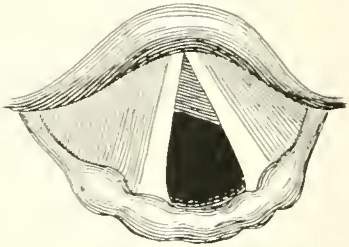

Fig. 82.

Fig. 81.—Illustrating schematically the inability of the cords to approximate in the center owing to disturbance in the cricothyroid muscle because of reflex interference with its innervation through the superior laryngeal nerve.

Fig. 82.—Illustrating schematically the inability of the posterior end of the cord to approximate its fellow because of interference with muscular innervation, through the inferior laryngeal nerve.

Tickling in the Larynx and Dry, Hacking Cough.—These are early reflex symptoms, the path of the reflex being through the laryngeal branches of the vagus. The inflammation in the lung stimulates the sensory fibers of the vagus and sends an impulse to the medulla which is transferred to the laryngeal branches of the same nerve.

Unfortunately these symptoms attract attention to the throat and away from the lungs. These unfortunate reflexes have cost many lives by misdirecting both the patient and the physician, the true source of the cough not being found until the disease had advanced to an incurable stage. Uvulas, tonsils, and adenoids have been removed, septums straightened, and pharyngeal catarrh treated for this symptom, when a chest examination would have shown that the chief trouble was below the larynx instead of above it. These reflex symptoms on the part of the larynx are most severe during periods of toxemia when the inflammation in the pulmonary process is acute and the peripheral ·irritation is exaggerated. Consequently, we would expect in early tuberculosis to find some of the toxic symptoms present when tickling in the larynx and cough are due to pulmonary tuberculosis. If not, some of Group III might be present to give us our clue to the localization of the disease.

Disturbance in Heart Action.—There are peculiar characteristic changes in pulse rate in early tuberculosis, both when toxemia is present and when absent. During the periods when the patient is suffering from toxemia sympathetic disturbances overbalance the parasympathetic in nearly all instances and the patient has unduly rapid heart action on exertion: but these periods of toxemia are not constant. Most of the time, the patient with early tuberculosis, is comparatively free from toxemia. Although free from toxemia, yet the heart action may be rapid. The central stimulation of the sympathetics from toxins and such conditions as fear, worry, discouragement and pain also produce an accelerating influence. The increased rate of metabolic change which is induced by the disease also calls for increased heart action. The rate of the pulse then is a resultant of many factors. At certain times, however, the direct nerve influence seems to be greatest. The antagonistic action between these two divisions of the vegetative nervous system is constantly present. When the patient is at rest and free from toxemia and all depressive states such as those of anxiety, discouragement, and disappointment, which produce general sympathetic stimulation and increased adrenin secretion, his pulse may be normal, or even below normal. Particularly is this true of the sthenic individual. In the hyposthenic, however, there is more apt to be increased rapidity. When the patient begins to exercise, however, then the pulse becomes more rapid than would be normal for the particular individual. It is further characteristic of the pulse that it does not return to its normal rate as quickly as it should after it has become accelerated. *The peculiar characteristics of the pulse in early tuberculosis are produced by a combination of stimulation of the sympathetics and adrenal and thyroid action on the one hand and reflex stimulation of the vagus on the other producing an instability the degree of which differs in different individuals and under different conditions.* It may be slow under certain conditions and rapid in others. It may be rapid in one individual and slow in others, as fully illustrated in Figs. 29, page 200, and 34, page 208.

Disturbances on the Part of the Digestive System.—Symptoms of reflex origin on the part of the digestive system are also variable. During periods of acute toxemia the inhibitory action of the sympathetic nervous system prevails in most instances, consequently the patient shows a lack of appetite, coated tongue, a deficiency of gastrointestinal juices and an inhibition of motility. In individuals in whom sympathetic tonus predominates over parasympathetic this may be more or less constant even during periods of quiescence. In those patients, however, of sthenic build, and in others in whom we might expect to find an underlying vagotonia, we are apt to have during periods of quiescence an increase in appetite, increase in the gastrointestinal sècretions and muscle tonus, even to the point of slowing of the intestinal contents and spasticity of the colon.

Loss of Weight.—This is more apt to occur as a result of toxemia than reflex action. It is when the sympathetics predominate over the parasympathetics that we have the greatest disturbance in the gastrointestinal tract followed by marked loss of weight; and this is most common when central sympathetic stimulation is marked as a result of toxemia. There is ample evidence, too, that a disturbed endocrine balance is an important factor in loss of weight.

Chest and Shoulder Pains.—A symptom which should receive more attention than is now given it is the reflex chest and shoulder pains. These present two characteristics. One of these, the reflex sensory disturbance, expresses itself in the muscles and superficial tissues supplied by cervical nerves and in the third to fifth thoracic zones as shown by Head (see Figs. 27 and 28, pages 182 and 183). This gives discomfort over the apices of the lungs, over the third to fifth interspaces anteriorly, and in the scapular region posteriorly. The other type of pain is that which seems to be a definite neuritis affecting the branches of the cervical nerves which take their origin from cells in the cervical segments of the cord which are adjacent to and in communication with other cells receiving afferent impulses, through the sympathetics, from the inflamed lung. This last type of pain requires for its production that the disease be an old one and that the irritation be kept up over a sufficiently long time to produce degenerative changes in the nerve itself. The sensory reflex, however, may be due to a new involvement or to activity in an old one. Patients often speak of these pains, particularly the shoulder pains, as rheumatism; and they are often so considered by the physician. They vary from a slight ache to a pain of severe degree, but are usually only moderately severe. In taking histories it is always well to ask the patient whether he has had rheumatism or neuralgia in the shoulders or chest and he will often reply that he has. It will frequently be found to involve the side where there is an old process. Pain is also frequently due to an inflammation of the intercostal nerves which extends from the inflamed and thickened pleura. This is probably a cause of apical pains in limited lesions.

Flushing of the Face.—This symptom is, as a rule, not present in truly early tuberculosis. It requires a certain amount of involvement before it is present. A feeling of heat in the cheek is complained of at times by patients with a fairly early process, but is usually confined to the periods when the disease is distinctly active. It is due to a reflex dilatation of the blood vessels of the face, head and ear. The irritation causing it in the face passes through the vagus to mediate with the fifth cranial; and in the ear through the sympathetics and the third cervical spinal nerve. It is confined to the side on which the activity is present, or, if both sides are involved, it is usually most marked on the side of the more active involvement.

SYMPTOMS DUE TO THE TUBERCULOUS PROCESS ITSELF.

There are but few symptoms which are due to the tuberculous process itself, but these are most distinctive of all symptoms present in early clinical tuberculosis. Unfortunately, they are somewhat later in appearing than some of those of the other groups. They usually presuppose either an involvement of some duration or one of considerable extent. In fact, when we make a diagnosis of clinical tuberculosis it has really existed for some time and there is usually a considerable area involved. The symptoms due to the tuberculous process itself should rarely be mistaken for any other disease or condition.

Frequent and Protracted Colds.—So-called frequent and protracted colds are, as a rule, tuberculous bronchitis. The patient complains of taking cold easily or of colds which hang on longer than they should. Inquiry will usually show that these are not the ordinary attacks of coryza. If they begin as head colds, they end up with a cough; but are usually due to a bronchitis from the beginning. Such attacks should always be viewed with suspicion; and, if accompanied by temperature, or other symptoms due to toxemia, or, by those belonging to the reflex group, should call for a careful examination of the chest. When these attacks are more severe, the patient often calls them "la grippe" and speaks of having one attack of "la grippe" after another. Repeated winter coughs are often of this type. One of the most important things to understand about tuberculosis is that none of the symptoms are necessarily constant. The toxic, or even the reflex symptoms following one of these definite outbreaks may soon pass away. Activity in the whole process may clear up and show no further symptoms for months, or even for years, yet be of a tuberculous nature. Symptoms disappear long before the process is pathologically healed.

Spitting of Blood.—This indicates tuberculosis in every instance unless it can be definitely proved otherwise. While there are other causes for this symptom, yet the fact that the patient raises blood is presumptive evidence that tuberculosis is present; and, the evidence is conclusive in most cases when the blood is bright and mixed with sputum. Pinkish colored saliva sometimes comes when the gums are bleeding; occasionally we find streaks of blood in the presence of bronchitis; bloody expectoration may come from heart lesions; but the clinician must know that these causes are rare in comparison with the one great cause of blood spitting,—tuberculosis. If the examiner will look for pulmonary tuberculosis in all such cases he will not go far wrong. If he cannot find the evidence himself it is his duty to make a probable diagnosis and call the assistance of some expert diagnostician. Vicarious menstruation is often spoken of. Personally, I have never seen spitting of blood at the menstrual time except in the tuberculous patient. Women who are tuberculous often spit blood at this time. *If there is one symptom of early tuberculosis that should always be interpreted in the interest of the patient, it is spitting of blood.* Blood spitting is not always preceded by other symptoms, nor is it always followed by them. It may be due to tuberculosis and yet be the only symptom present. This would occur where an old inactive lesion had ruptured, causing laceration of a small blood vessel, and then healed at once without producing any extension of the disease. Blood spitting is more accurate in diagnosis than the Wassermann reaction.

Pleurisy.—Pleurisy is another symptom which should not be mistaken. We have been grossly negligent of this symptom in the past. Pleurisy has a definite meaning. In a very large per cent of cases it is subpleural tuberculous. *It is evidence of active tuberculosis.* The profession must learn that tuberculosis of the pleura is as serious as tuberculosis of any other organ, with the exception that it is limited in extent. If properly regarded, it offers excellent chances for cure. *Pleurisy is a metastasis from some other focus in the body which must be active or the metastasis would not occur; consequently it should be treated seriously.* If the fact that there is an involvement in the pleura is not of sufficient consequence in the mind of the practitioner, the fact that this is the extension from some other focus which is active or the extension could not have occurred, should be of sufficient consequence to cause this symptom to be treated seriously. This symptom should make a diagnosis of itself unless it can be definitely proved that the pleurisy has some other cause. This applies to dry pleurisy as well as to pleurisy with effusion. As a rule, however, symptoms in Group I will manifest themselves during this time and some of those in Group II, particularly the laryngeal symptoms, will usually be present.

Sputum.—While sputum is not a common accompaniment of early tuberculosis yet it is present more often than we suspect. Whenever the pathological process is extending, it causes an exudation in the adjacent tissues and there may be a slight amount of sputum present. At first this may be only mucus, but, sooner or later, ulceration and caseation of small tubercles may occur and bacillus-bearing sputum may be found. Sputum should always be examined. It is best to give the patient a bottle and require him to bring all the sputum which he raises for twenty-four, forty-eight, or even seventy-two hours. This sputum should be treated, not only according to the ordinary methods of examination, but by antiformin or digestion and homogenization, as described in Chapter XX. If this course is carefully followed one will often be surprised at finding bacilli when unsuspected. The inexactness of basing a diagnosis upon the examination of a single sample can be understood if we realize that, while the patient may expectorate six or eight times a day he may not expectorate bacilli more than once in the entire twenty-four hours, or once in two or three days; hence, the examination of a sample, unless bacilli are found, is worthless. Where bacilli are not found the lymphocyte count should be noted. In tuberculous sputum there is often a high lymphocyte count. If 40 or 50 per cent of lymphocytes should be found it should be considered suspicious of tuberculosis. The albumin reaction as described on page 538, Vol. 1, is of considerable value when carefully made; but since we also find it present in lung abscess, bronchiectasis and the infected bronchi following influenza, it must be interpreted with care in these postinfluenzal days.

RELATIVE VALUE OF THE VARIOUS GROUPS OF SYMPTOMS.

From the preceding discussion it will be seen that of all the symptoms which are associated with early tuberculosis, there are only the four in Group III which point directly and definitely to the lung. By understanding the causes underlying these three groups of symptoms we can nearly always pick out one or more belonging to each group when early active tuberculosis is present. This combination will give us sufficient evidence on which to base a probable diagnosis; and when considered along with the physical signs which are present there should be comparatively little doubt left in the examiner's mind as to whether early clinical tuberculosis is or is not present. This subject is discussed more fully in Chapter XXI.

CHAPTER XV.

THE DIAGNOSIS OF EARLY PULMONARY TUBERCULOSIS: PHYSICAL EXAMINATION OF THE PATIENT.

General Considerations.—There is no best method of examining a patient that is applicable to all examiners. Different men work in different ways. One will develop a peculiar phase of physical examination so that he comes to rely on it, while another will rely most on another. One will follow some particular method and become wedded to it, while another will utterly fail in its application. So we are led to believe that it is not wholly the method of the examiner, but rather his knowledge of conditions and the manner in which he interprets his data, the judgment which he uses, that makes for a correct diagnosis.

Favorable Conditions for Making Examination Important.—The diagnosis of early clinical tuberculosis depends on little things. The symptoms, for the most part are not severe and would be considered unimportant were they not attributable to so serious a disease. The changes in physical signs are often so slight that they are only appreciable upon careful painstaking observation. This makes it essential that the conditions under which an examination is to be made should be as favorable as possible.

The patient should be examined in a warm room so that he will be comfortable and relaxed. The difference between examining through relaxed and rigid tissues must be considered in all instances and will be dwelt upon more as our discussion proceeds.

Nervousness on the part of the patient should be allayed if possible. The patient should be bare to the waist so as to permit of unobstructed observation of the chest; and the waist band should be loose so as not to interfere with free motion of the chest wall. Inspection, palpation, percussion and auscultation are all practiced with greater ease and greater precision when the chest is bare.

Whenever possible the patient should be examined in a sitting posture. The percussion note is different when the patient is lying down from what it is when he is sitting up, because we not only percuss the chest but the couch, bed or stool that the patient is sitting or lying on, as I shall describe later. The auscultatory sounds also differ when the patient lies down and sits up because the type of breathing changes. The sounds at the apex are accentuated when the patient is examined in a sitting posture. Another reason why the sitting posture is preferable is because the examiner is more comfortable and can make a better examination. Difficulties are much increased by bending down over a patient in bed. It is best for the patient to sit on a stool without a back, the seat being a few inches higher than the stool of the examiner. This permits a favorable view of the entire chest and particularly of the motion of the bases. The patient should face the light. Daylight is preferable to artificial light. The differences in the musculature and soft tissues can be determined very much better and it always seems to me that I can note the changes in motion better with natural than artificial light. Whether this is because of the natural light being more intense or whether the fact that it is a white light while artificial light is nearer the tones of the skin, I am not sure.

Methodical Examination Necessary.—Early tuberculosis may be overlooked by not examining methodically. There are certain parts of the chest which should be examined more carefully than others but every portion should be included in the examination. Each examiner should develop for himself a certain routine which includes a complete investigation of the appearance of the patient as a whole, and the chest in particular; the condition of both the soft parts and bony thorax; the movements on respiration; the resistance of the chest both on palpation and percussion; the alterations in pitch and in the quality of the note, and the variations in auscultatory findings. The heart should always be included in an examination for pulmonary tuberculosis.

The apices, being portions of the lungs which are usually affected when our advice is sought, should be examined most carefully. If nothing is found in the apices, although we are led by the clinical history to believe that a tuberculous infection is present, we must not forget the possibility of hilus infection. The bronchial glands are usually harbingers of the early infections and it is not uncommon to find an extension of the disease out into the adjacent pulmonary tissue. The interscapular area above the level of the sixth dorsal vertebra and the areas along the sternum and border of the heart should be carefully examined in such cases. In fact, one must be ready for any surprise, as far as localization is concerned, for the bacilli might find lodgment and start an active process in any portion of the lung. The difficulties in making a satisfactory physical examination of the tissues near the hilus must be borne in mind, and the x-ray should be utilized when possible.

Negative evidence on physical examination should not be too hastily interpreted as showing absence of disease, for it may be only proof of our inability to find the infection.

Physician's Duty in Suspected Cases.—In case physical examination fails to reveal a lesion and yet the examiner believes from the clinical history that tuberculosis is present, it is his duty to tell the patient frankly what he suspects so that he may regulate his life accordingly. It is not necessary to frighten him. In fact, this should be avoided. When early tuberculosis is present or when it is suspected is the time when the physician has a great responsibility upon him; for, depending on the manner in which he handles the situation, hangs the life and usefulness of the patient. The shock of learning of a suspected infection can be taken away by telling the patient of the hopeful side of the disease. It is well to state that it is only neglected tuberculosis that kills, but that early lesions will usually heal, providing the patient faces the situation bravely and cooperates in the treatment.

Etiological Classification of the Changes Found on Physical Examination.—The subjective symptoms in early tuberculosis may be divided according to their etiology into those due to toxemia; those of reflex origin; and those due to the tuberculous process *per se,* as mentioned in the preceding chapter. The physical signs may be divided according to their etiology into two groups; those of reflex origin, often called objective symptoms, and those due to the tuberculous process *per se,* as follows:

PHYSICAL SIGNS IN EARLY TUBERCULOSIS

OF REFLEX ORIGIN	DUE TO TUBERCULOUS PROCESS *per se**
Dilatation of the pupil. Interference with approximation of vocal cords. Lagging of chest wall, regional and general. Rigidity of muscles (spasm).	Increased density of tissue on palpation. Increased resistance to finger on percussion. Alteration in quality and pitch of percussion note. Alteration in inspiratory and expiratory rhythm. Alteration in strength, quality and pitch of respiratory sounds. Adventitious sounds (râles).

*All physical signs due to the tuberculous process *per se* are modified by whatever changes are found in the soft parts and bony thorax. The amount of subcutaneous tissue and muscle substance covering the chest must be considered; so must the condition of these tissues, whether the subcutaneous tissue is in normal amount and of normal elastic texture or atrophied; and whether the muscles are normal in size and texture or in a state of increased tone (spasm), or in a state of atrophy.

Factors Causing Changes on Inspection, Palpation, Percussion and Auscultation.—The careful diagnostician arrives at his diagnosis by a process of reasoning. The methods of deriving data on physical examination of the chest follow the laws of physics, and he who can interpret these best will be most successful in physical diagnosis.

When we make a physical examination of the chest of an individual suffering from clinical tuberculosis we are attempting to determine the physical changes which have occurred as a result of the infection, both directly and remotely. These changes are made evident to us through our senses of sight, touch and hearing and are determined by inspection, palpation, percussion and auscultation. They manifest themselves not only in alterations of the pulmonary tissues, but in changes of the soft tissues covering the bony thorax and in the bony thorax itself.

The physical examination for early clinical tuberculosis was long considered as being a process of determining the departures from the normal in sounds heard on percussion and auscultation, as a result of the tuberculous infiltration in the lung. Practically everything was excluded from the mind except the thickening of the lung tissue and the way this affected the percussion and auscultatory note, although the change in vocal fremitus was noted by some. Later it was found that this thickening of the lung tissue was also perceptible to the fingers during percussion, and that there was a deficiency in the respiratory movements over the apex in which the infiltration was situated. But this conception is still too restricted.

Our attention has been centered too much on the changes in the tissue *per se* and not enough on the surrounding structures. The elements which make up the sensation conveyed to the fingers on palpating or percussing the chest and those which are conveyed to the ear on percussing and auscultating the chest are several in number; the infiltration itself; the tissues surrounding it, pulmonary, cardiac, mediastinal, and pleural; the tissues covering the bony cage, and the cage itself. The tissues within the thorax are often influenced by changes which have resulted from previous or other present pathological conditions. The percussion note, particularly if heavy stroke is used, is also greatly influenced by the patient's surroundings, whether he is sitting on a soft bed or a solid stool or standing on a solid floor. Palpation, percussion and auscultation are influenced markedly by the thickness and tone of subcutaneous tissue and muscles.

Factors Which Affect Soft Structures Covering the Bony Thorax.—The findings obtained on physical examination differ greatly according to the amount of soft tissues covering the bony thorax, whether the muscles are large or small and whether there is a great or small amount of subcutaneous tissue. It also differs according to the density of these tissues; therefore, according to whether the muscles are in a state of contraction (spasm) or relaxation, whether the muscles are of normal texture or degenerated. The writer first called attention to the diagnostic importance of the fact that the muscles covering the chest show a reflex regional contraction (spasm) in the presence of acute inflammation in the lung and that both muscles and subcutaneous tissue degenerate if the inflammation assumes a chronic course, in 1909.[1] These changes greatly modify the data obtained on physical examination and must be taken into consideration in their interpretation. They also offer valuable signs of themselves, which, when interpreted properly, will give valuable hints of present or past inflammatory processes within the lung. The soft tissues are also influenced by occupation. The muscles of the right side of the chest, particularly the pectoralis, trapezius, levator anguli scapulæ, and rhomboidei, at times, show an hypertrophy because of extra work. *As a rule, however, these muscles except the rhomboidei and levator scapula are smaller than on the left.* A general reduction in size and lengthening of the muscles of the shoulder girdle permitting the shoulder to drop, is usually found as a result of righthandedness. The changes in the soft structures, whether physiological or pathological must be taken into consideration in making a physical examination. A point of differential value is that muscles, degenerated pathologically, show atrophy of the segmentally corresponding subcutaneous tissue, while those lengthened by right or lefthandedness and those degenerated because of disuse do not; also, the sternocleidomastoideus is not apt to be affected by either of these conditions, although the clavicular portion lengthens some on the side of greater use. Further, the total density of the lung tissue is greater if the change is due to pathological thickening. This is determined by increased palpatory or percussion resistance.

Cause of Reflex Spasm and Degeneration of Soft Tissues.—The reflex which affects the soft tissues covering the bony thorax in the presence of inflammation of the pulmonary tissue is produced in the same manner as that which affects the muscles of the abdomen when the abdominal viscera are inflamed. This reflex is best known in appendicitis but is present in all inflammatory conditions affecting the internal viscera.

Could we but trace the reflex, there is no doubt but that inflammation of every internal organ would be expressed on some portion of the surface of the body by motor, sensory and, if it persists for a sufficient time, by trophic changes.

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[1] A New Physical Sign Found in the Presence of Inflammatory Conditions of the Lungs and Pleura, Journal American Medical Association, March 6, 1909; and Muscle Spasm and Degeneration in Intrathoracic Inflammations and Light Touch Palpation, C. V. Mosby Co., St. Louis, 1912.

The path of these reflexes is for the most part through the sympathetics to the cord; although there may be other paths such as through the vagus and spinal accessory to the sternocleidomastoideus and trapezius muscles.

The reflex is segmental in character. This point must be clearly understood, otherwise the peculiar distribution of the reflex will not be appreciated. The meaning of this will be clear by recalling that a spinal nerve is made up of many individual fibers which take their origin from individual cells in the cord. The cells giving origin to the fibers which go to make up a nerve trunk may be distributed through more than one segment of the cord, but individual cells, when irritated, transmit their impulses only through the fibers to which they give origin. It is not necessary that the entire nerve be involved in the reflex, the stimulation may be confined to a few or it may involve many of its fibers. Only those fibers are involved which take their origin from cells which lie adjacent to and receive irritation from other nerve cells which give origin to fibers, particularly sympathetic fibers, which receive impulses from the viscera. This is why a portion of a muscle or one of a group may show the motor and trophic changes when the entire muscle or entire group are supplied by the same nerve. The path of this reflex is shown in Plates III and IV.

Example of the Effect of Spasm and Degeneration.—A concrete example of the way this reflex acts is as follows. Suppose an individual is suffering from an active tuberculous infiltration of the left apex. The lung, being supplied by sympathetic fibers which originate in the upper five or six thoracic segments of the cord, when inflamed sends impulses back to the cord over the sensory sympathetics whose cell bodies lie in the posterior root ganglia corresponding to these segments. Here the impulses are transmitted to other neurons which carry them upward in the cord to the cervical segments and there transmit them to the cervical motor and sensory nerves which take their origin from these segments. The cervical nerves which transmit the pulmonary reflexes most readily are apparently the third, fourth, and fifth, although we find reflexes in all, at times. The nerves which take their origin from the seg-

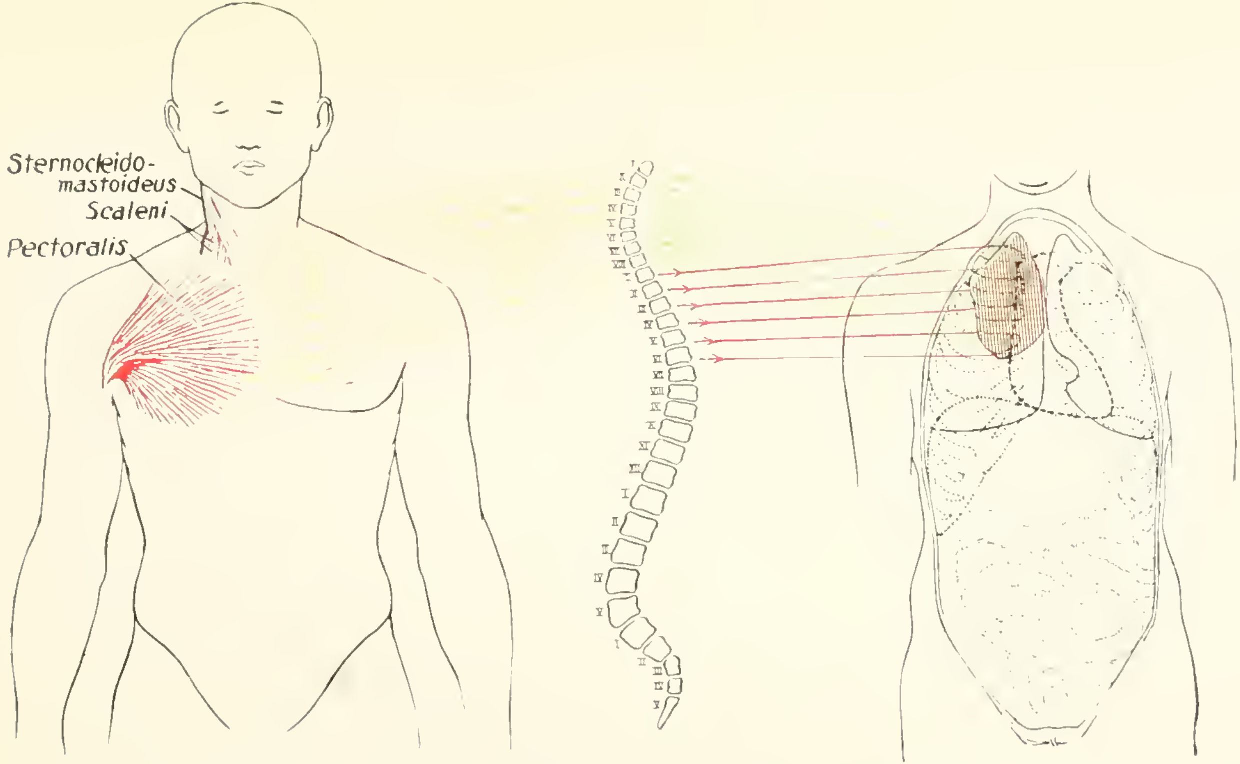

PLATE III.—SHOWING SCHEMATICALLY THE PATHS OF THE NERVOUS IMPULSES WHICH CAUSE THE REFLEX SPASM AND DEGENERATION OF MUSCLES WHEN PULMONARY TISSUE IS INFLAMED (ANTERIOR VIEW). THE DIAPHRAGM BEING INVISIBLE IS OMITTED IN THIS CUT.

The *red lines* represent the sympathetic innervation of the lung arising from the upper six thoracic segments. The *green lines* represent the sensory impulses carried from the inflamed lung through the sympathetics to the cervical portion of the cord. The *yellow lines* represent the cervical motor nerves which complete the reflex, carrying the impulses from the cervical portion of the cord to the muscles represented in red. The muscles of greatest interest from a diagnostic standpoint, are the sternocleidomastoideus, the scalenus anticus and medius, and the pectoralis, which are in spasm when the lung is acutely inflamed, and degenerated when the inflammation becomes chronic. These muscles are shown in *red* in the illustration. The *arrows* indicate the direction in which impulses travel.

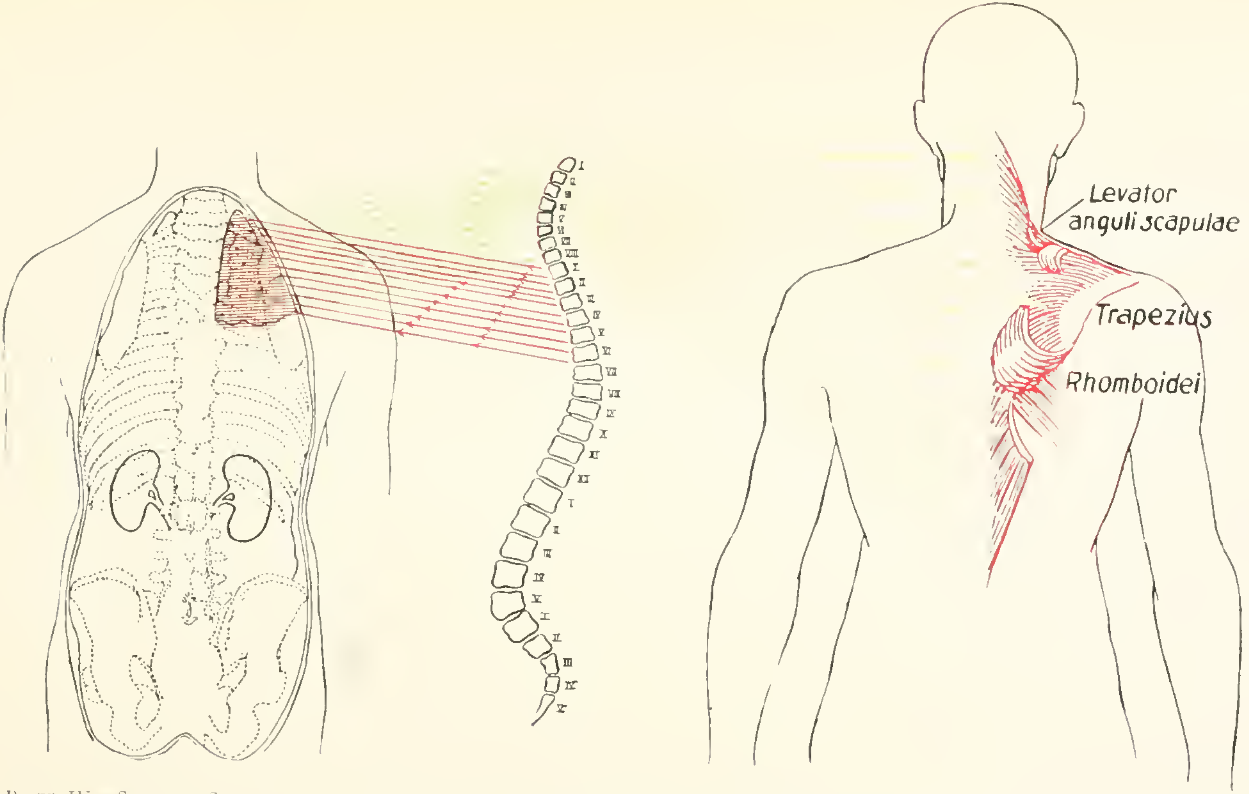

Plate IV. Showing Schematically the Paths of the Nervous Impulses Which Cause the Reflex Spasm and Degeneration of Muscles, When Pulmonary Tissue is Inflamed (Posterior View). The Diaphragm Being Invisible Is Omitted in this Cut.

The *red lines* represent the sympathetic innervation of the lung arising from the upper six thoracic segments. The *green lines* represent the sensory impulses carried from the inflamed lung through the sympathetics to the cervical portion of the cord. The *yellow lines* represent the cervical motor nerves which complete the reflex, carrying the impulses from the cervical portion of the cord to the muscles represented in *red*. The muscles of greatest interest from a diagnostic standpoint on the posterior surface of the chest, are the trapezius, levator anguli scapulæ, and rhomboidei, which are in spasm when the pulmonary tissue is acutely inflamed and degenerated when the inflammation becomes chronic. The *arrows* indicate the direction in which impulses travel.

ments of the cord which receive the impulses from the lung, supply the surface of the neck and upper chest with sensation, the muscles of the shoulder girdle, the diaphragm, and part of the arm with motor power, and all soft tissues of the upper thorax with trophic impulses. While the inflammation is acute, only sensory and motor changes are apparent, but after it has existed for a prolonged time the trophic changes in the soft tissues are also apparent. The two changes which interest us most from a diagnostic standpoint are the motor and trophic. In the example here given the motor reflex would show in a lagging of the left side of the chest owing to the motor disturbance of the diaphragm through the left phrenic nerve; and an increased tension (spasm) of the muscles covering the left apex, particularly the sternocleidomastoideus, scaleni, upper fibers of the pectoralis, trapezius, levator anguli scapulæ and rhomboidei. The degree of contraction (spasm) differs according to the extent and activity of the lesion because the wider the area of inflammation the greater the number of sympathetic fibers and the greater the number of cells in the cord that are irritated; and, the more active the inflammation, the greater the number of stimuli. Should this process go on to a chronic state or finally heal, then the trophic changes show themselves in a wasting of the soft parts. The skin, subcutaneous tissue, and muscles which are in the area of the reflex, degenerate, and show this change by a thinning of the skin and subcutaneous tissue, a general wasting of the muscle substance, and a feeling of doughiness. Should the activity continue, or should it again appear after the process has become chronic, then the muscles, degenerated because of the chronic process, would also show an attempt at contraction (spasm) because of the renewed inflammation.

A second path of reflex action to the sternocleidomastoideus and trapezius is through the spinal accessorius, the neurons of which also receive irritation from their connection with sensory fibers in the vagus. The double path to these muscles may account for the fact that the reflex spasm is usually marked in these two muscles.

No one who desires to be accurate in diagnosis can fail to consider these changes in the soft structures covering the bony thorax; nor must he fail to recognize that the findings over a rigid thoracic cage are different on palpation, percussion and auscultation, from those over one of normal elasticity.

With this introduction, we are now able to take up the discussion of the individual methods of examination and the data obtained by them with a greater degree of accuracy than heretofore. While many difficulties still present, particularly on the part of the examiner, yet some of the sources of gross error from the standpoint of the patient are eliminated. Inspection and palpation at once assume a greater importance in diagnosis; and, percussion and auscultation become more accurate.

INSPECTION.

Inspection has hitherto not been given its full value in the diagnosis of diseases within the chest. If one will but look at the chest, observing alterations in the subcutaneous tissue and muscles and then carefully observe the motion of the chest wall, and alterations in the contour of the chest itself, he will have important data upon which to base an opinion of the nature of any pathological process, either active or inactive, affecting the tissues within. Some of these changes in old chronic affections have been partly, but not fully, recognized for a long time. The changes in early clinical tuberculosis, however, are not so well known, but are no less valuable in the information which they furnish.

In order to be able to detect the abnormal, we must have a definite picture of the normal thorax in mind. As has been definitely shown in Chapter XIII, by the drawings from Mills, there is no absolutely fixed type of chest. Nevertheless, we must look upon the flattened chest as an evidence of physical deterioration, and make the normal rotund chest our ideal. At the same time, we must not call departures from this type pathological.

Dilatation of the Pupil.—Dilatation of the pupil on the affected side is not an uncommon sign in pulmonary tuberculosis. This dilatation comes from stimulation of the sympathetics which

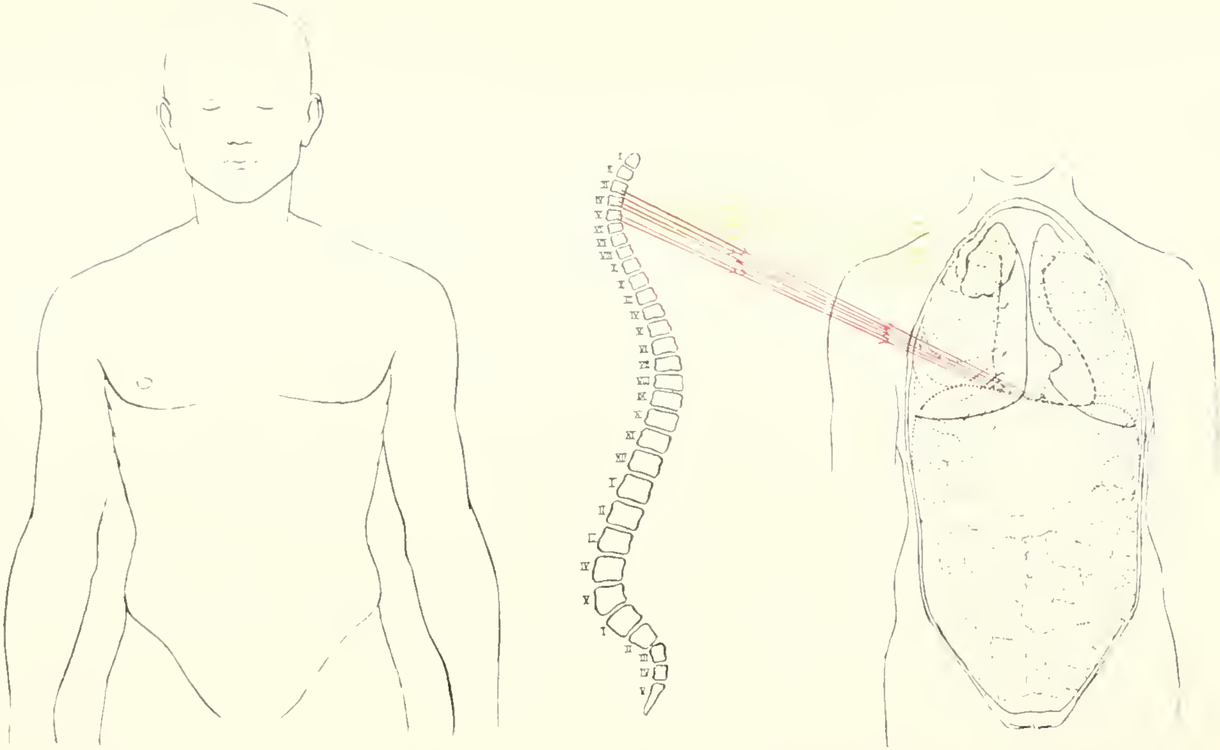

Plate V. Showing Schematically the Motor Disturbance of the Central Tendon of the Diaphragm, Resulting from Inflammation in the Lung.

The *green lines* represent the sensory impulses carried from the inflamed area in the lung to the IIId, IVth, and Vth cervical segments of the cord. The *red lines* represent the phrenic nerves carrying the reflex motor impulses from the IIId, IVth, and Vth segments of the cord to the central tendon of the diaphragm. The *arrows* indicate the direction in which impulses travel.

take their origin from the second to fourth thoracic segments of the cord. It will be recalled that the pupil is innervated by both the parasympathetic and sympathetic divisions of the vegetative system. The parasympathetic contracts, the sympathetic dilates the pupil, as described more fully on page 198. This dilatation is evidence of the sympathetic overcoming the parasympathetic tonus. This sign is inconstant. It may be present in one individual and not in another. It may be present at one time and absent at another. The same is true of symptoms and signs in all organs where we have this double innervation and reflex stimulation of filaments of both systems. The same inconstancy is noted in the heart action and in the digestive disturbances, as described elsewhere.

Lagging and Diminished Motion.—Lagging and diminished motion may affect any part of the chest wall. It has usually been described as affecting the apex alone, but we find it also affecting the base, and, in fact, the entire side as will be appreciated from Fig. 60, in which it is shown schematically that the motion of the entire thorax is lessened, and from Plate V, in which the diaphragm reflex from the lung is schematically illustrated.

Lagging, as a sign in early tuberculosis, has at least three elements which enter into its etiology; a reflex motor disturbance affecting the muscles of respiration, particularly those covering the apex and the diaphragm, a diminished elasticity of the pulmonary tissue and pleuritis. Diminished elasticity, however, cannot be a very important factor in the early lesions. Its most marked action comes later in the disease. Diminished motion may be a sign of previous or present inflammation of the pleura, the lagging being due to inhibited motion caused by acute inflammation, or adhesions, or contractions. It can also be produced by pericarditis and inflammations below the diaphragm.

In order to determine lagging, the chest should be bared of clothing so that the movements of the entire thorax are visible and unobstructed. The movement of the lower border of the ribs is as important as that of the apices, consequently the waist should not be constricted by clothing.

Diminished motion may often be detected more readily by palpation than by inspection, although practice will usually enable one to see it. If one is inspecting the apices for it, he can do so either by seating himself before the patient and fixing his eyes so as to watch the motion of both sides at once; or, he may stand back of the seated patient and look down over the front of both apices at the same time. To detect this sign at the base, it is best to sit facing the patient who sits on a stool a little higher than the examiner. Diminished motion may be more readily seen by placing the hands on the apices or bases as shown in Figs. 83 and 84. Whether this acts mainly by aiding vision or more by adding the factor of feeling to inspection does not seem wholly clear.

When inspecting a chest for diminished motion the patient should first breathe normally, for forced breathing will often overcome the lessened motion. After natural breathing, then deep breathing should be observed. Some observers claim that several forced inspirations will tire the affected muscles and exaggerate diminished motion; it seems to me, on the contrary, that it more often overcomes it. If diminished motion of one apex or base is found in suspected early pulmonary tuberculosis, it points to the probable lesion being on that side, providing acute and chronic inflammation of the pleura be eliminated.

If distinct signs of active tuberculosis are found in one apex and the motion of both sides of the chest is the same, it points strongly to both lungs being affected, unless a recent or old pleurisy is present at the base opposite the well defined lesion. If one apex is the seat of a fairly extensive lesion which shows slight or moderate activity, while the other apex is the seat of a fresh, though less extensive infiltration, the greatest diminution of motion is apt to be on the side of the newer infection, because the new reflex is more marked. When diminished motion is present on both sides it furnishes confusing information, but this can usually be clarified by the changes observed in the subcutaneous tissues and muscles. Percussion and auscultation should leave no doubt as to its interpretation. Close observation and experience will also tell one about what degree of motion to expect in a given chest.

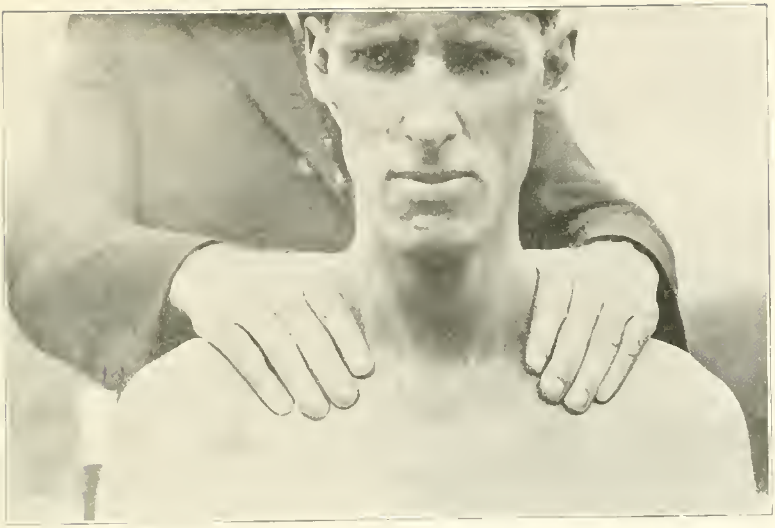

Fig. 83.—Illustrating the method of detecting diminished motion at the apices.

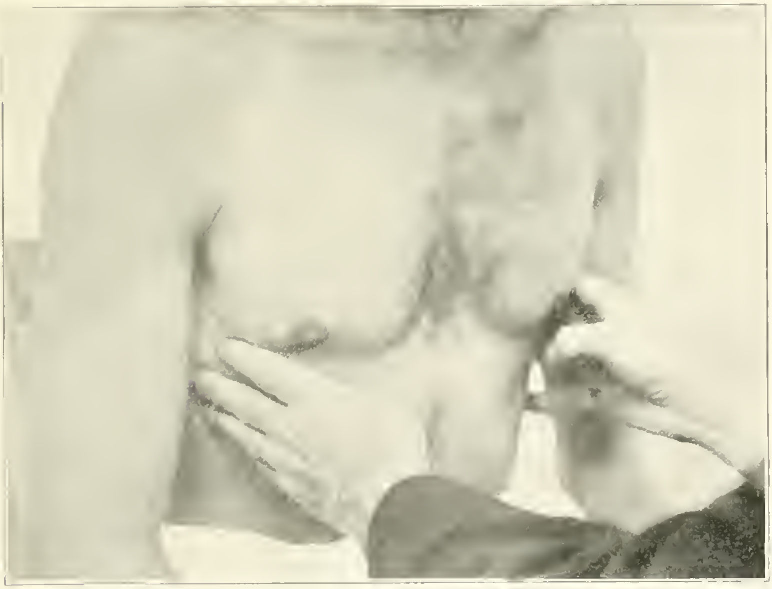

Fig. 84.—Illustrating the method of detecting diminished motion at the base. The hands should be laid gently on the chest so as not to interfere with the normal respiratory movements. Often a pressure no greater than one or two pounds will completely check the movements of the ribs.

The path of the motor reflex causing lagging or diminished motion of the chest wall, is from the inflamed area in the lung through the sympathetic fibers to the cord, upward to the cervical portion of the cord and back through the motor nerve filaments to the diaphragm and other respiratory muscles supplied by cervical nerves; also through the pulmonary vagus to the medulla, and out through the spinal accessory to the sternocleidomastoideus and trapezius as previously mentioned.

State of Muscles and Subcutaneous Tissues.—Valuable information can be obtained in examining for early tuberculosis by carefully observing the condition of the muscles and subcutaneous tissue. It is necessary in this connection to emphasize the importance of examiners familiarizing themselves with the normal condition of the soft parts covering the chest so that they may be able to detect departures from the normal. In individuals of moderate or much flesh, the neck should be fairly full and there should be no distinct groove above the clavicle but one below on the side of the hand used most. In patients who are thin, on the other hand, the clavicle and neck muscles will be naturally more or less prominent and the supraspinous fossa may be somewhat flattened. In the child these parts on the two sides are symmetrical, but, as the individual grows older, an asymmetry gradually develops. When there has been a chronic inflammation of an apex followed by contraction, the soft tissues not only atrophy, but they are drawn down toward the apex, a condition which makes the neck muscles, particularly the heads of the sternocleidomastoideus, and sometimes other neck muscles, unusually prominent, as shown in Fig. 95, *A* and *B*, page 466.

Occupational and Pathological Changes in Soft Parts Covering Apex.—The departures from the normal condition are partly occupational and partly pathological. The *occupational changes* in the soft parts of the neck and upper chest consist in an asymmetrical condition of the muscles which control the motions of the arm. The muscles of the right side are usually developed to a greater extent than those of the left *in those who use the arm much*, because of the condition of right-handedness which is almost universal. *In those who use them less*, the muscles become smaller, lengthen, and permit the shoulder to drop, as is evidenced on the right side in right-handedness, and on the left side in left-handedness. This dropping of the shoulder carries with it the insertion of the pectoralis muscle, draws the upper fibers away from the clavicle, causing the infraclavicular cupping which is often mistaken for shrinking of the apex.

The *pathological changes* are for the most part based on reflex motor and trophic disturbances which result from acute or chronic inflammations within the lung, the most common cause being tuberculosis. They consist of:

1. An increased prominence of the muscles, particularly the sternocleidomastoideus, scaleni, trapezius and levator anguii scapulæ, often seen when active inflammation is present in an apex which has not previously been involved. Sometimes this cannot be determined by inspection, but only by palpation.

2. A diminution in size of the muscles and a wasting of the subcutaneous tissue when a lesion is present of a chronic nature or when one has been present at some previous time.

3. An increased prominence of some of the bundles of the muscles which appear somewhat wasted, and a wasting of the subcutaneous tissue in corresponding sensory zones when we have an active inflammation in an area which had previously been the seat of a chronic process. This cannot always be determined by inspection but must sometimes be left to palpation.

The importance of recognizing these changes in the soft parts is apparent when we realize the frequency with which the pulmonary apices are affected by tuberculosis in adults. As previously mentioned, of 400 autopsies made by Hart, the apices were affected in 254 or 63.4 per cent. In the cases reported by Naegeli 71.43 per cent showed apical involvement. This means that some time in the life of many of the 63.4 per cent of Hart's cases and 71.43 per cent of Naegeli's cases there were probably reflex motor changes (spasm) in the neck muscles which could have been recognized on inspection and palpation had they been examined. It further means that reflex trophic changes leading to degeneration, which were a result of the chronic tuberculous infection of the underlying apex, were present in the muscles and subcutaneous tissue covering the apices in about the same percentage of cases. These motor and trophic changes differ in degree but must be taken into consideration in making physical examinations of chests for they of themselves alter the findings often to a very large degree; and, in many instances the trophic change will call attention to the presence of old chronic pathological processes within the lung which are large enough to alter the resistance on palpation and the data derived by percussion and auscultation far more than the recent process itself.

It is also desirable to determine, if possible, whether these apical lesions when present are healed or whether they are in an active state at the time of the examination. Of all methods of physical examination this can be best judged by inspection and palpation, by noting the motion of the chest wall and whether or not the muscles covering the apex have an increased or decreased tone. By auscultation we can sometimes judge whether or not activity is present; but there are no sounds heard on auscultation which always mean activity. Harsh breathing, rough breathing, diminished breathing, prolonged expiration and even râles may be present in arrested tuberculosis. Diminished motion, barring pleuritis and fibrosis, suggests some active interference with the muscles of respiration, the sternocleidomastoideus, and particularly the scaleni and diaphragm on that side; and if there is an increased tension of the apical muscles on the same side, we have strong evidence of there being an active inflammatory process in the corresponding lung. If, along with these motor changes there is a positive tuberculous history, we are justified in considering that there is an active lesion present. If, on the other hand, we find evidence of a lesion on palpation, percussion and auscultation, but can find no alteration in motion of the suspected side and no increased tension of the apical muscles; but, on the contrary, a decreased tension and wasting of the subcutaneous tissue, together with an absence of symptoms, we are justified in considering that we are dealing with an old lesion which is now quiescent or healed.

Changes in Contour of Trapezius Muscle.—A significant sign of an old chronic inflammatory process in the lung, evident on inspection is a change in the contour of that portion of the trapezius muscle formed by the junction of the upper or occipital portion with the middle portion. The normal trapezius gradually curves from the acromium to the upper cervical and occipital portions. When the underlying apex has been the seat of a chronic inflammatory process such as tuberculosis, the muscle degenerates and the subcutaneous tissue over it becomes thinner than is found on the opposite side, as shown in Fig. 85, *A* and *B*. The middle portion flattens and the junction between the middle and the superior fibers assumes more of an angular appearance than normal and this junction appears at a considerably lower level than the curve on the opposite side. The portion above the angle also appears smaller than its fellow on the other side. This condition is present in those cases which show a narrowing of the apical isthmus (Kroenig). It is not a sign of active or clinical tuberculosis, but an indication of a process which has been there long enough to produce chronic changes. Difficulty in assigning proper diagnostic value to the changes in the trapezius arises because of similar changes found on the side of the hand used most.

These degenerative changes in the trapezius are particularly well shown in Fig. 95, *A* and *B*, page 466. This patient suffered from chronic tuberculosis affecting both lungs. The disease on the right side, however, was much more extensive and had produced much more destruction of tissue than that on the left side. The reflex trophic changes in the muscles and subcutaneous tissue is well illustrated in the neck and shoulder girdle on both sides, particularly on the right. The right sternocleidomastoideus is much smaller than the left one. The same is true of the pectoralis, trapezius, levator anguli scapulæ and rhomboidei. The wasting of these muscles has permitted the clavicle, scapula, and shoulder as a whole to drop down and fall away from its central moorings. It will also be noticed that as a result of the wasting of the soft tissues and their contraction the supraclavicular notches are deeper than normal and the two heads of the sternocleidomastoidei stand out very prominently. The influence which these muscle changes exert upon percussion of

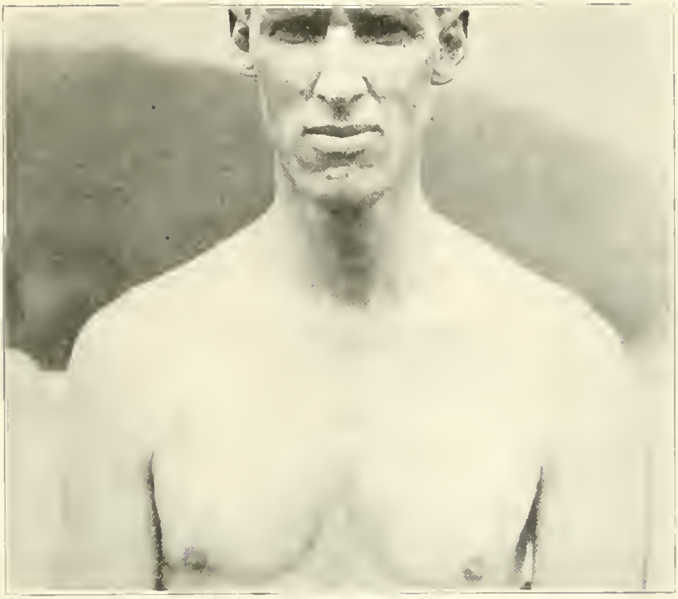

Fig. 85*A*.

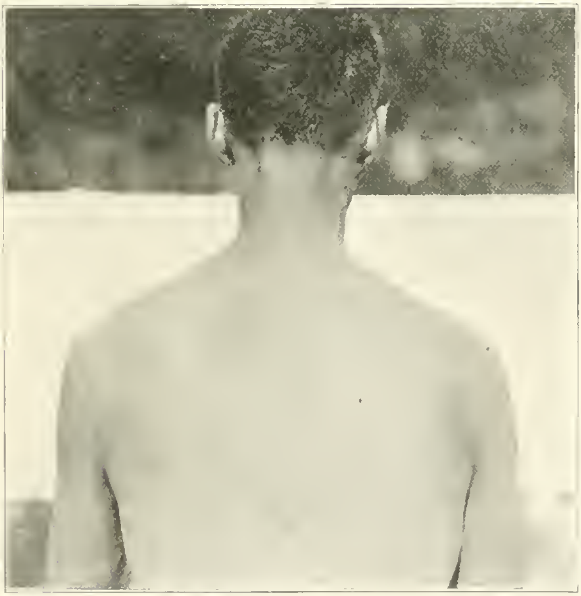

Fig. 85*B*.

Fig. 85.—Illustrating the change in the contour of the trapezius muscle. *A*, anteriorly; *B*, posteriorly. This patient has suffered from a chronic lesion in the right lung for many years, which has resulted in a marked degeneration of the muscles on that side, which accentuates the changes due to right handedness.

the apices may be further inferred from Figs. 96, *A* and *B*, and 97, *A* and *B*, page 468. The latter are taken from Brecke's Article in "Handbuch der Tuberkulose."

This sign is well worth observing in early tuberculosis now that we know so many of our cases are the result of renewed activity in old lesions or extensions from the other apex. It assists in the proper interpretation of our findings, and becomes of special value if accompanied on the same side by lagging and increased density of the pulmonary tissues as determined by palpation and percussion. It may also be due to occupational changes but this cause can be eliminated as mentioned above.

Mammary Gland.—The mammary gland is often found to be smaller on the side where there has been an old chronic or healed lesion. This is true of non-tuberculous as well as tuberculous processes. This shows as a decrease in the subcutaneous tissue and probably also in a decrease in the size of the gland itself. Not only does the gland appear smaller than the one on the other side but it usually hangs lower on the chest wall, on account of the atrophy of the tissues which support it. This condition is also noted on the side of the arm used more.

The conditions above mentioned are the most important ones to be observed on inspection in examining for early tuberculosis; but one should also note the general appearance of the patient, the condition of his skin, any departure from the normal such as enlarged lymphatic glands, evidence of endocrine disturbances, and inequality of the pupils.

PALPATION.

What Can Be Determined by Palpation.—In a former discussion of this subject[2] the writer said: "If we were to confine our examinations simply to the detection of fremitus, palpation would not be of much value in early tuberculosis, but palpation in its broader sense consists in deriving by the sense of touch whatever evidence we can of the presence of tuberculosis." Today, the writer is able to say that in his own practice, palpation, and particularly palpation aided by inspection, has assumed an importance much greater than percussion in the diagnosis and interpretation of intrathoracic disease.

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[2] Pulmonary Tuberculosis, William Wood & Co., New York, 1908.

By palpation we determine the following data useful in the diagnosis of early tuberculosis:

1. Whether the muscles are normal; or whether they show an increased tone (spasm) indicating either an occupational hypertrophy or a reflex hypertonicity caused by an inflammatory process within the thorax; or, whether they are smaller and lengthened as on the side of the hand used more, or show a reflex trophic disturbance caused by some intrathoracic inflammatory condition which has assumed a chronic course.

2. Whether the skin is normal or whether it shows regional trophic change with loss of elasticity.

3. Whether the subcutaneous tissue is normal or whether it is degenerated. The only common cause of a regional degeneration of the subcutaneous tissue covering the neck and chest known to the writer is that of reflex trophic disturbance caused by a chronic inflammatory process within the thorax.

4. Increased resistance resulting from intrapulmonary infiltrations. In fact, palpation will give him who is expert in its use nearly all information that can be obtained on percussion, and some that cannot. Infiltrations, cavity, emphysema, thickened pleura, pleural effusions, mediastinal growths, the position of the heart, the lower border of the lungs, the upper border of the apices the position of the liver, and the outline of the stomach under certain conditions can all be determined by it.

5. The presence or absence of diminished motion from which we infer either that the muscles of respiration, particularly the scaleni and diaphragm, are reflexly interfered with or are normal in their action.

6. The presence or absence of tactile fremitus.

7. The presence or absence of enlarged lymphatic glands.

Regional Spasm of Muscles.—By palpating the muscles of the neck and thorax we gain information additional to that obtained on inspection. The muscles which show reflex motor disturbance (spasm) most markedly in early active tuberculosis are the sternocleidomastoideus, scaleni, upper fibers of the pector-

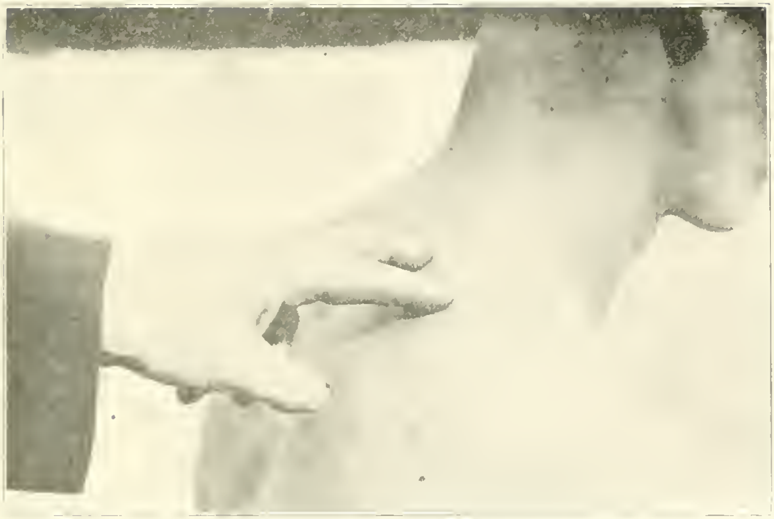

Fig. 86.—Illustrating the method of palpating to determine the condition of the muscles and other soft tissues. The muscles should be in the natural position of repose and the touch should be light, scarcely causing an indentation of the tissues.

alis, trapezius, levator anguli scapulæ, rhomboidei and diaphragm, all of which are palpable except the last. Interference with the diaphragm is inferred by lagging of the side, when pleurisy is excluded.

It is characteristic of this motor change that it is regional and not general in character, affecting at times only a portion of a muscle or a part of a group of muscles.

Such signs and symptoms as dilatation of the pupil, and the disturbance in the pulse rate, and those on the part of the secretory and motor functions of the gastrointestinal canal are inconstant because they are reflexes in which both parasympathetic and sympathetic divisions of the vegetative system are implicated. Their action being antagonistic, sometimes parasympathetic tonus predominates, at other times sympathetic. The reflex changes in the muscles (spasm), on the other hand, are constant, the increased tone appearing when the inflammation begins and continuing until healing has taken place.

The normal muscle has a distinctly elastic feel. The entire muscle seems to have the same general tone. When in repose the texture is firm, but not tense. When in normal contraction the entire muscle takes on an increased tension. *Whether in repose or contraction it is elastic.*

When in reflex contraction (spasm) some of the muscle fibers are in a state of increased tone, although all may not be.

The reflex spasm is present as an early sign of intrapulmonary inflammation, hence is of value as a sign of early active tuberculosis.

This contraction of the muscles may often be determined by inspection, but palpation shows the increased tone better and gives more definite information. The method of palpating is shown in Fig. 86.

The reflex contraction may be determined best, by *light touch* palpation. If the finger tips are placed on the muscles in a manner similar to the way they are placed on the piano keys, and the examiner will make a rapid vibratory motion pressing and releasing, he will feel a distinct rebound on the part of the contracting fibers like that offered by a bow string when treated in the same manner. This "bow string" feeling is quite characteristic. This increased tone may also be determined by gently feeling the muscle or by picking it up when possible and rolling it between the thumb and finger.

When this spasm is present, together with a suspicious clinical history, we may be almost certain that an active tuberculous lesion lies within the corresponding lung. Figs. 61 to 64, pages 330 to 332, illustrate schematically the principal neck muscles involved in the reflex from the lung.

This spasm is sometimes present in muscles which have degenerated because of a previous inflammatory process such as when we have a renewed activity in an old quiescent focus. Under such conditions the subcutaneous tissue is also wasted. The contraction (spasm) under these circumstances as a rule, is more feeble than in the undegenerated muscle, owing to the atrophy present, and the muscle has a doughy lifeless feeling instead of its normal elasticity.

Increased tone (spasm) of the neck and chest muscles is constant in early active tuberculosis. This was first pointed out by the writer but has been confirmed by many others. St. Galecki[3] in a recent contribution reports the finding of spasm in 93 per cent of early cases examined.

Regional Atrophy of Skin, Muscles, and Subcutaneous Tissue.—A regional reflex atrophy affecting the skin, muscles and subcutaneous tissues is present over certain areas of the chest when an old chronic inflammatory process has affected the underlying portion of the lung. The wasting is evident to the eye, but it is determined better by palpation. The parts which are particularly affected are the same muscles that have been enumerated as showing spasm, and the subcutaneous tissue and skin above the second rib anteriorly and the spine of the scapula posteriorly. Degeneration in the interscapular region indicates old extrapulmonary hilus inflammation.

Atrophy of the muscles shows by a wasting of their substance, the belly of the muscles being smaller than normal. The substance between the bundles disappears and the muscles lose their rotundity and appear as stringy masses. The tips of the fingers can be more easily pressed between the bundles than when normal. The muscles as a whole show a lack of elasticity and present a distinctly doughy resistance to the finger.

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[3] Die Inspektion und die Palpation des Thorax in der Diagnose der Lungentuberkulose, Brauer's Beitrage zur Klinik der Tuberkulose, Bd. xxx, 1914.

The wasting of the subcutaneous tissue caused by the lung shows in the supraclavicular and infraclavicular notches, and supraspinous fossæ, from the hilus, in the interscapular region. It can best be determined by comparing with the normal side; or, where both sides are involved, with the subcutaneous tissue over other parts of the chest. By simply pressing or by picking up the skin and subcutaneous tissue between the thumb and fingers, it will be seen at once that on the side of the atrophy the tissue is much thinner than on the other side. This atrophy affects the skin also, and in some instances, can be readily determined. Sometimes the line of demarcation between the atrophied and normal subcutaneous tissue is quite abrupt, at other times gradual.

A point of differential value in distinguishing between atrophy following muscle disuse and reflex trophic disturbance is that in the former the subcutaneous tissue should not be affected, while in the latter it is also atrophied.

These localized atrophies should always make one think of the possibility of some chronic inflammatory process, particularly an old chronic tuberculous process, on the corresponding side. If interscapular, it indicates that the extrapulmonary hilus glands have sometime in the past been chronically inflamed.

Determining of Pulmonary Infiltrations by Palpation.—It is possible to learn to recognize differences in density of tissue by touch as well as by percussion. I have found it practical to use palpation in the examination of the chest where percussion is generally used. Examiners have been gradually replacing the interpretation of the sound emitted on stroke by the feel of resistance noted by the fingers. The utilization of touch to determine this resistance is only carrying the point a little further. It has one great advantage. One can palpate a definite small area; but when he percusses, according to the strength of the stroke, he causes, as previously mentioned, the entire chest, in fact the entire body, and even the stool or bed on which the patient sits or lies, to be thrown into vibration. After he becomes proficient the examiner will notice that a different sensation is conveyed to the palpating finger by the different organs and different pathological conditions. Solid and hollow viscera are readily differentiated. Infiltrations in the lung are easily distinguished from normal pulmonary tissue. Emphysema, thickened pleura, pleural effusions, and pulmonary cavities may all be detected by certain characteristic sensations conveyed to the palpating finger. The thickened pleura is accompanied by a peculiar doughy or edematous condition of the overlying soft parts indicative of degeneration (see Chapter XXV, Volume II).

Palpation and percussion are usually considered to be distinctly different procedures and if we interpret the percussion findings entirely according to sound, they are so; but, when we interpret them according to the resistance to the finger it can readily be understood that the difference is one of degree. Both palpation and percussion disturb the equilibrium of and set up vibrations in, the tissues palpated or percussed. These vibrations differ according to the nature of and particularly according to the density of the tissues through which they pass. The sensations conveyed to the palpating finger or the finger used as a pleximeter will also differ according to the density of the tissues. Thus we learn to interpret a feeling of increased resistance to the finger as meaning increased density of the tissues. That we can determine the differences between substances of different density by touch can easily be perceived by palpating, lightly, pieces of metal or wood of different thicknesses. The marked difference in a piece of wood one-quarter of an inch thick and another piece one or two inches thick can be perceived by almost anyone. Differences of lesser degree, while more difficult to determine, can be easily made out with practice.

For the beginner, in palpating chests for the differences in resistance, I would suggest that the resistance over the heart in the fourth interspace to the left of the sternum be compared with that over the lung in the same interspace beyond the border of the heart, or that the resistance over the liver be compared with that over the lung above it.

Patience and perseverence are required to master palpation the same as percussion and auscultation; but he who will take the time and exercise the patience to learn it, will be rewarded by having at his command a method of examining which is free from that error which comes from the widespread vibrations set up by percussion, and one which will often greatly aid him in forming an opinion. *Tactus eruditus* is a factor in examining by light touch palpation, but practice and a determination to learn it will make any intelligent examiner proficient. *The practitioner who has studied percussion and auscultation for four years while in college, and practiced it from one to twenty years since, must not expect to take up palpation over night. It requires practice to become proficient in its use.*

When palpating a chest for an early apical lesion, it is well to begin below and systematically feel the resistance over the chest until the apex is reached. It has long been recognized that there is an increased resistance to the pleximeter finger and also slightly higher pitched note in percussing from below upward when the upper interspaces are reached. This also holds for the area immediately above the clavicle. This is most probably partly due to the fact that we are percussing over muscles near their insertion, which condition offers more resistance in some instances than when we are percussing over the middle of the muscle; partly due to the fact that the first interspace is narrower than the others and partly to the relatively less lung tissue and greater total thickness of the muscles as the apex is approached (see Fig. 65, page 334). This same condition is to be noted on palpation, and must not be confused with an increased resistance which might be due to a pathological thickening of the underlying pulmonary tissues. It is extremely important to bear these points in mind so as to avoid errors in diagnosis.

Palpation is found particularly valuable in the supraclavicular notch. *We often notice a certain increased resistance (hardness) over this area, when the underlying apex is the seat of a tuberculous process, which, if once appreciated, will not be easily forgotten and which carries with it considerable diagnostic importance.*

After several years' experience in the use of palpation for the determination of areas of different tissue density, I can recommend it as being practical where the difference is great and as being possible where it is small. He who attempts to take it up for the first time may find many discouragements, but this is true of any method. Were it taught to medical students the difficulties would be little if any greater than those experienced on percussion. Comparatively few men can percuss and interpret their findings accurately. The advantage of having palpation and percussion to check each other cannot be ignored. I would especially urge those who are giving special attention to the diagnosis of diseases within the chest and abdomen to practice palpation, particularly light touch palpation with the same earnestness that they now practice percussion.

Diminished Motion. This sign was described when discussing inspection. It can be determined more readily, however, by palpation than by inspection. Sitting before the patient, who should preferably sit on a stool a little higher than the examiner, one should place his hands over the lower ribs in the mid-axillary lines while the patient breathes quietly, then deeply (forced breathing often overcomes a slight or moderate lagging). If either base lags it can be readily determined. The apices may be examined in the same way, but it is preferable to examine them while the examiner stands behind the patient, with his thumbs over the supraspinous fossæ and his fingers extending down over the clavicles. The pressure exerted by the palpating hands must be very gentle and be the same with both hands. Even a pressure of one or two pounds may be sufficient to overcome the motion of the chest wall. Diminished motion may show posteriorly as well as anteriorly.

Lagging is not difficult to detect when one side only is involved; but when bilateral involvement is present there is some difficulty because the movements may be about equal on the two sides. When there is a double lesion, however, it is usually more marked on one side than the other. It can plainly be seen that there should be a lagging on the side of the pronounced involvement, so a limited motion on the other is presumptive evidence that a lesion exists in that lung also. It must not be forgotten that the left base naturally shows a slightly lessened motion when compared with the right, probably because the right crus is larger and the right side of the diaphragm has the liver for a fulcrum which acts in forcing the lower part of the chest wall out.

Tactile Fremitus.—Tactile fremitus is the vibratory sensation conveyed to the palpating finger or hand when laid upon the chest while the patient is speaking. Certain definite words, like "one," "two," "three," or "ninety-nine," are used because they produce a maximum vibratory effect.

Fremitus differs according to the character of the voice and the character and thickness of the tissues through which the vibrations must pass. It is naturally more marked over the right apex. The reason for this has probably been correctly assigned by Fetterolf to the fact that the trachea lies in direct contact with the pulmonary tissue on the right while the large vessels lie beween the trachea and pulmonary tissue on the left.

Any pathological condition which produces a thickening of the pulmonary tissue will cause an increased fremitus; therefore this should be of value in the diagnosis of tuberculosis. There are so many signs of greater value, however, that this is rarely relied on to any extent. Vocal fremitus, usually spoken of as voice transmission, as determined by the stethoscope is more reliable than that determined by palpation.

Enlarged Glands.—The determination of enlarged glands, particularly those of the cervical group at times has some bearing on the pulmonary involvement.

PERCUSSION.

Light or Heavy Percussion.—During recent years methods of examination have taken on many improvements. In percussion, as previously stated, there is a growing tendency toward a lighter stroke and greater attention to the sensation of resistance conveyed to the finger in comparison with the sound produced by the stroke.

It was believed until recently that it was necessary to employ a heavy stroke in order to percuss deep lying organs or portions of organs, but we now know this is erroneous. This idea is illustrated in Fig. 87 taken from Sahli's work on Physical Diagnosis. Deep infiltrations in the lung, the deep borders of the heart and liver, and mediastinal growths can be determined by light touch palpation. The lighter the percussion stroke the better, because a heavy stroke causes widespread vibrations of all the neighboring tissues as well as the bony thorax; and causes increased difficulty in interpretation. A stroke so light that it is scarcely audible can be perceived through the entire thickness of the chest, as can readily be determined by placing the palm of the hand on the front of the chest wall while a light percussion stroke is applied to the back.

Careful diagnosticians have learned that there are many sources of error in the interpretation of the percussion note and are more and more depending on the resistance noted by the finger. This is of special importance in all chest conditions. There are many methods of percussion but there are none superior to finger-finger percussion if the examiner is skilled in its use; for it has the advantage of delivering the stroke and receiving the impression of the stroke by instruments in which the sense of touch may be keenly cultivated. The best percussion, however, is that done in the manner which the particular examiner knows best and interprets most accurately.

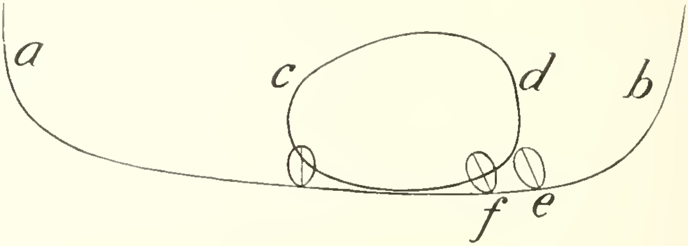

Fig. 87.—The acoustic sphere of action of the blow in deep percussion. Origin of the deep dullness. An illustration purporting to show the supposed necessity for heavy percussion. *a-b*, cross-section through the anterior wall of the thorax; *c-d*, cross-section through the heart; elliptical shadow areas representing the sphere of vibration caused by percussion; *c-d*, the shadow area. is wholly within the lung and does not reach the heart; *f*, is partly in lung tissue and partly in the heart, consequently it gives information. (Sahli.) As explained throughout these pages, this conception is erroneous. The deep borders of the heart may be outlined by a touch or by a stroke so light that it is barely audible.

It is very important in comparing the findings on percussion over different areas of the chest, to percuss under as nearly the

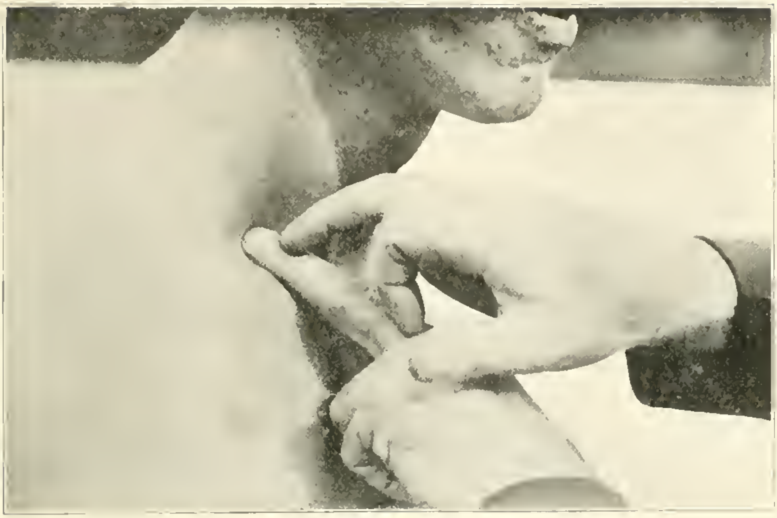

Fig. 88*A*.

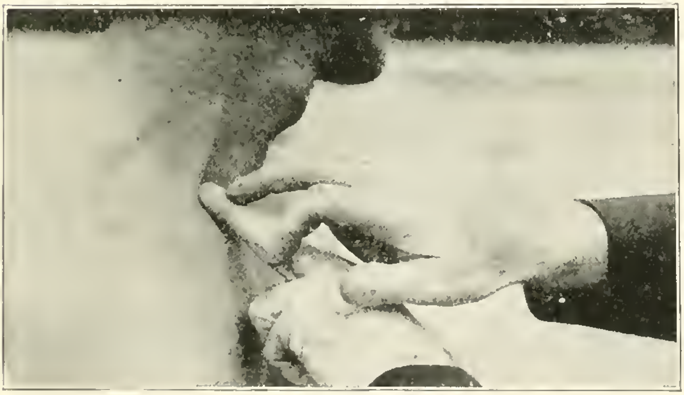

Fig. 88*B*.

Fig. 88.—Illustrating a common error in percussing the apices. *A*, proper position, showing percussion of the apices while the patient's head is erect and tension removed from the sternocleidomastoideus and other neck muscles. *B*, wrong position, percussing same when the head is turned and bent over toward the opposite side, thus putting the sternocleidomastoideus and other muscles on tension, thereby raising the pitch of the percussion note and increasing the resistance to the percussion finger.

same conditions as possible. The arms should be in the same position. *All muscles should be relaxed. The head should be in the median position.* A very common error is to percuss one supraclavicular notch with the head erect and face forward and the other with the face turned to one or the other side; or trying to compare findings derived while the head is turned to one side, as shown in Fig. 88, *A* and *B*. Turning the face to the opposite side throws the muscles of the neck on a stretch which results in a higher pitched note and greater resistance to the finger, thus giving wrong information. The important muscles which affect apical percussion anteriorly are shown schematically in Fig. 89.

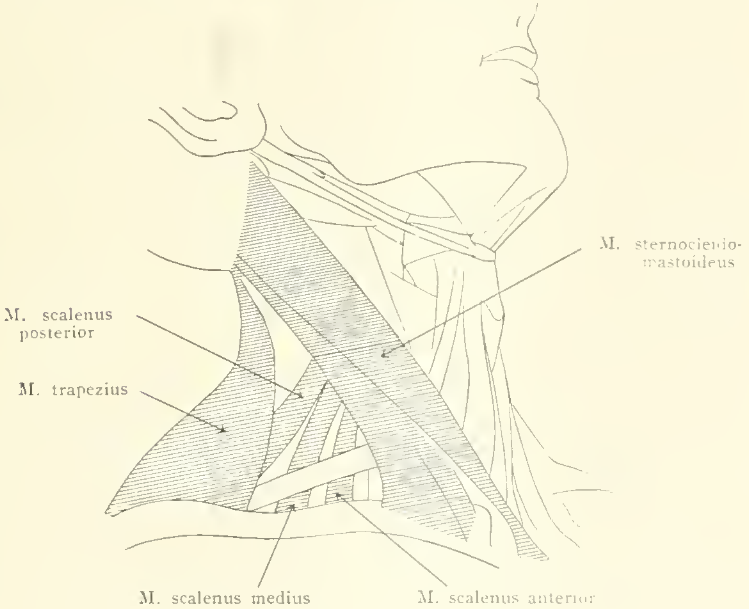

Fig. 89.—Important muscles which affect percussion of apices anteriorly, shown schematically. (Gerhartz.)

The percussion note differs in different chests. Each must be considered by itself, consequently there is no note or tone that can be interpreted to mean a definite pathological condition. Increased or decreased resistance to the finger, however, always means relatively greater or lesser density of tissue.

Percussion Changes in Early Clinical Tuberculosis.—The percussion findings in early tuberculosis vary greatly in different individuals, and are modified by the nature of the infiltration itself; by the condition of the lung tissue and surrounding structures; by the elasticity of the bony thorax; by the amount of soft tissue covering the bony thorax and by the condition of the muscles and subcutaneous tissue. One will soon learn to gauge the allowance to be made on account of the soft structures.

If the infiltration is dense this should give a high pitch to the note emitted on percussion and a marked resistance to the pleximeter finger. If it is scattered it might cause so slight an elevation of the pitch as not to be noticeable; it might take on a tympanitic quality, or it might cause so little increased density of the tissues that it would not be perceived by the palpating finger. This is what we would find if we were percussing a lung containing a scattered infection, without regarding its coverings. But we find it necessary to take the bony frame work and soft tissues covering the parts into consideration. This increases our difficulties of interpretation and throws greater responsibility upon the examiner.

Recalling the statistics of Hart and Naegeli before mentioned, which show that between 60 and 70 per cent of adults have tuberculous deposits of greater or lesser dimensions in their lungs, usually the apices, we can see that we are not always percussing fresh infiltrations of normal lung tissue. Recalling also that these old chronic processes cause trophic disturbances in the soft structures covering the apices (muscles and subcutaneous tissue) then we have an idea of the problem before us. The greater the amount of solid tissue covering the portion of the lungs through which we percuss and the greater the density of such tissue, the higher the pitch of the note and the greater the resistance to the finger. Therefore, large muscles and muscles in contraction and a large amount of firm subcutaneous tissue cause higher pitch and increased finger resistance as compared with the same muscle in relaxation, and the same subcutaneous tissue when atrophied.

Bearing these factors in mind we find the following different conditions on percussion of the apices when tuberculous involvement is present.

1. *A primary active involvement of one apex.* This condition presents normal subcutaneous tissue over both apices, normal muscles over the healthy apex and muscles showing increased tone (spasm) over the diseased apex. The amount of spasm present depends on the extent and degree of activity in the lesion. Of course, occupational influences must be considered.

In this instance it is necessary to compare the percussion of a normal lung through normal tissue, with an infiltrated lung through muscles whose tone is increased by being thrown into reflex spasm. The result is that the pitch of the percussion note and the resistance to the finger are increased by both factors over the diseased apex, and should be readily detected. Often, if not generally, the changes wrought in the percussion phenomena by the increased tone of the muscles is greater in early clinical tuberculosis than that produced by the pathological thickening in the pulmonary tissue. The effect of the scaleni in fixing the 1st and 2nd ribs is especially marked.

2. *An old quiescent or healed lesion in one apex.* According to the extent of the lesion we find degeneration of the apical muscles, particularly the sternocleidomastoideus, the trapezius, also the upper fibers of the pectoralis, the three scaleni, the levator anguli scapulæ and rhomboidei. The subcutaneous tissue over this area is also atrophied. The points where this wasting can be best detected are in the supraclavicular and infraclavicular regions, and with less ease in the supraspinous fossa. This condition is illustrated schematically by Fig. 90, page 422.

In this instance it is necessary to compare percussion findings through atrophied muscles and subcutaneous tissue over one apex with normal elastic muscles and normal subcutaneous tissue over the other. If the pulmonary tissue at the two apices could be the same under these conditions, the percussion note would be higher and the resistance to the finger greater on the side of the normal soft parts because of the greater thickness of the soft tissues on that side. This same holds true when one apex is the seat of a tuberculous infiltration unless there is a sufficient amount of pathological thickening present in the lung to more than overcome the decrease in soft tissues which is due to wasting of the muscles and subcutaneous tissue. If the lesion in this instance has thoroughly healed there should be no clinical symptoms present, the muscles should show no extra tension barring pleural adhesions, the motion of the diaphragm should show no marked diminution and the percussion over the remaining portion of the lung should be practically normal. Theoretically, at least, there is a slight change in the percussion note when diminished motion is present because the lung contains relatively less air.

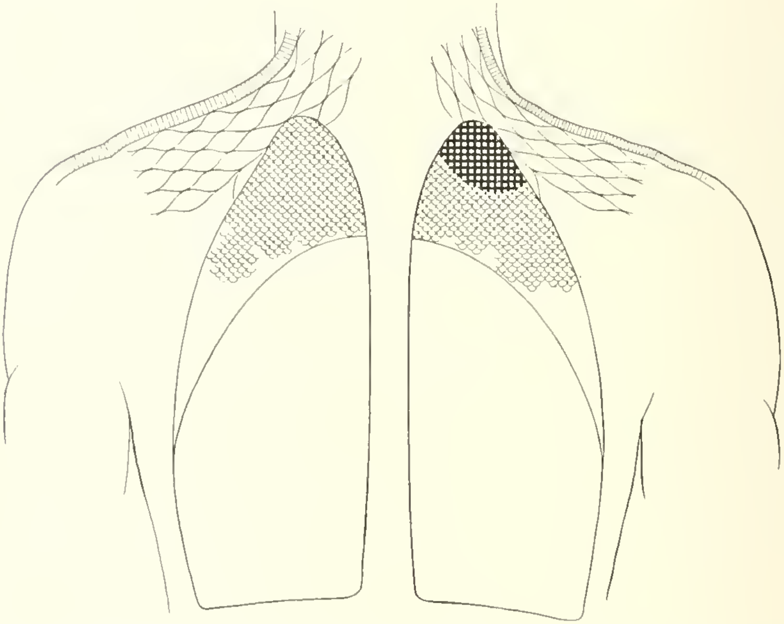

Fig. 90.—Illustrating schematically the degenerative effects upon soft tissues produced reflexly by an old chronic inflammation in the apex of the lung. It will be noticed that on the side where heavy shading indicates a lesion in the apex, the subcutaneous tissue and muscle mass is much smaller than on the other. This materially influences the percussion note, and must be considered in interpreting findings, on palpation, percussion, and auscultation.

Percussion over the affected apex under these circumstances may show an increased resistance to the finger and a higher pitched note than the normal apex, or one equal in intensity or one less in intensity according to the amount of thickening present in the lung and the amount of wasting which has taken place in the muscles and subcutaneous tissues. The greatest changes occur in patients with heavy muscles and thick subcutaneous tissue. Not infrequently is the higher pitch and greater resistance found on the normal side.

3. *An old quiescent focus in one apex which has taken upon itself renewed activity.* Here we have the same degeneration of the muscles and subcutaneous tissue as mentioned above. Aside from this there is a renewed inflammation in the lung which causes the same muscles to contract reflexly (muscle spasm).

On percussion we are comparing the results obtained by percussing an infiltration through atrophied subcutaneous tissue and atrophied muscles, which are again thrown into a state of mild contraction, with those obtained by percussing a normal apex through normal muscles and normal subcutaneous tissue. It is impossible to tell by percussion what part of the changes found over the pathological apex is caused by the old lesion and what part is due to the new. Activity should be suspected in all such cases if diminished motion is present and the muscles should seem somewhat tense, especially if a positive clinical history is present. The percussion note, however, will be, at least theoretically, a little higher in pitch and the resistance to the finger will be somewhat greater for the amount of pathological thickening present, than is found over a healed lesion of equal extent because of the added factor of the slight increase in muscle tone. We meet the same conditions here, however, as we did above, and unless the amount of infiltration in the pulmonary tissue, plus the added factor of the contraction of the muscles is more than sufficient to compensate for the loss of subcutaneous tissue and wasting of the muscles, the percussion note and the resistance to the finger will be the greater over the healthy apex. This condition is often met with and very frequently misinterpreted. Here also, percussion findings may be greater, less, or equal to those of the healthy side according to the size of the infiltration and the amount of degeneration which has taken place in the muscles and subcutaneous tissues.

4. *An old healed focus in one apex and a new involvement of the other apex.* This presents a puzzling condition on percus-

sion. At one apex we have the atrophied muscles and subcutaneous tissue tending to decrease the percussion findings and a thickening of the pulmonary tissue due to the old quiescent focus, tending to increase the percussion findings; while at the other apex we have an increased tone in the muscles (spasm) and the new infiltration both tending to increase percussion findings.

If the findings on percussion of the two apices approach each other, the examiner may fall into the error of believing that both are normal. If the old lesion is of sufficient magnitude to give a higher pitched note and greater resistance to the finger on percussion than those obtained over the apex which is the seat of the new lesion, the new lesion is apt to be overlooked. If the findings on the side of the new lesion are markedly greater than over the apex with the old lesion, then the lesion is apt to be assigned to the side of the new lesion and the old one be overlooked.

Diminished motion of the side of the new involvement and spasm of the neck muscles should aid in the proper interpretation of the findings.

5. *An old quiescent focus which has again become the seat of activity in one apex and a new active process in the other apex.* This condition presents wasting of the muscles and subcutaneous tissue, with increased tone of the degenerated muscle (spasm) caused reflexly by the renewed activity, over the apex which is the seat of the old lesion; and an increased tone of the muscles (spasm) which may be slight or very marked, according to the size and activity of the lesion, over the apex of more recent involvement.

The percussion findings under these circumstances will depend very much upon the degree of infiltration present in the two lungs. If the findings over the two apices approach each other they may be erroneously considered as negative. If they greatly preponderate on one side the lesion may be assigned to that side alone.

In this class of cases the spasm of the muscles and lagging when considered together should help greatly. Both sides should show lagging because both apices are the seat of active disease.

It should be greater on the side of the new lesion unless the extent and activity is markedly greater on the other side. If the other side be the seat of a recent pleurisy or of pleuritic adhesions; or if both sides lag, the motion being about equal, then the condition of the neck muscles will give much information. A marked spasm of the neck muscles on the side of the new involvement with lagging equal or nearly equal on the two sides, indicates that activity and probably greater activity is present to cause the marked lagging on that side.

Percussion under these conditions should give definite findings, both sides showing plainly the departure from the normal. How much the increased pitch and increased resistance to the finger is due to the increased resistance offered by the contracted muscles, and how much to the infiltration *per se.* is difficult to determine; but, however this is, this type of involvement should be most readily made out on percussion.

Percussion does not tell that the lesion is active, but the spasm of the muscles, the changes on auscultation, and the positive clinical history should be present and aid in forming an opinion.

Conditions Within the Chest Which Alter the Percussion Note.—All pathological conditions in the lung, mediastinum, or pleural cavity alter the percussion findings. Of these, aside from previous tuberculous infections which have been discussed, fibrosis, resulting from an old pneumonia, emphysema, enlarged mediastinal glands and the results of pleurisy or empyema, are of special importance.

Where one lung has been seriously crippled by an extensive fibrosis, a pleurisy, empyema, or a tuberculous infiltration, the other lung takes upon itself a compensatory emphysema. The signs of a new tuberculous infection on this side are easily overlooked because the lung is comparatively less dense than normal. A percussion note over this emphysematous lung, plus the infiltration, may still be lower in pitch than that produced by normal lung tissue, and the resistance to the finger may be less than normal. Especial difficulty often presents in diagnosing a new infection when one lung has been the seat of a severe tuberculous involvement and the other after taking upon itself a high degree of compensatory emphysema, has become the seat of an infiltration.

Percussion Gives No Evidence of Activity.—From what precedes, it can be seen that percussion *per se* gives no information whatsoever that will enable us to differentiate between active, and quiescent or healed lesions; but should be considered in connection with the clinical history, state of the muscles, subcutaneous tissue, the action of the diaphragm and the tuberculin test. With our present knowledge of the frequency of quiescent and healed lesions in the lung, it is not sufficient to find evidence of pathological thickening on percussion. A presumptive diagnosis, even, cannot be given on this alone, for it might be due to an entirely harmless process which has fully spent its activity.

Kroenig's Apical Percussion.—This consists in mapping out the width of pulmonary resonance over the apex. Too much reliance has been placed on this in early diagnosis, as can be readily understood by recalling the pathological data which have been previously given. A narrowing of the area of apical resonance indicates only the presence of a pathological condition within that apex (usually tuberculosis) but does not indicate that it is the cause of clinical symptoms at the time. This must be determined by other methods such as the clinical history and the state of the muscles, the movement of the diaphragm, the auscultatory signs and the tuberculin test. Of itself, however, it gives very suggestive evidence.

AUSCULTATION.

This subject is one of the most important in connection with the early diagnosis of tuberculosis. It is likewise the object of more abuse than any other method. Comparatively few men have had sufficient training or experience to enable them to make a diagnosis of early clinical tuberculosis from the data derived by auscultation alone, before the advent of gross signs. With the more intensive teaching of medical students today this criticism should not hold with the newer generation of physicians. Unfortunately, the difficulties surrounding auscultation are not sufficiently recognized, and many men who examine chests, finding nothing which they recognize as abnormal, give an opinion accordingly in the face of definite symptoms of active disease. This often proves disastrous to the patient for it affords false security.

The physician who is not expert in examining chests must not make his diagnosis depend upon auscultation or upon any other single method of examination. If he does he will diagnose only late tuberculosis. He must rely more and more on clinical history and if not quite sure, should make a diagnosis only of *probable pulmonary tuberculosis;* but this should assure safety to the patient for he can then be told of the suspected condition and be put on proper treatment; or an expert can be called to complete the diagnosis.

Stethoscope.—There is a great difference in the various stethoscopes used by different examiners. All are not equally good. Some exaggerate sounds more than others. Those having much metal about them, particularly in the conducting portions, have a roar about them that is not present when the conducting tubes are of soft rubber. The small bell with soft rubber conducting tubes, transmits the sounds with the least change and has many advantages. The best stethoscope, however, is the one the individual examiner knows how to interpret best. The intensity of sound depends somewhat on the pressure applied to the stethoscope. The more lightly the bell of the instrument is applied to the chest the more intense the sound. Experimenting with the Bock stethoscope which has a diaphragm through which the sound passes, by which its intensity may be measured, I found that there was 25 per cent greater intensity in sound when the bell was applied lightly than when firm pressure was made.

No clothing should intervene between the stethoscope and the skin. When the chest is covered with hair great uncertainty exists in the study of the respiratory murmur and in the interpretation of any adventitious sounds that may be present. Different writers have recommended that the hairs be wet with water or greased with vaseline in order to overcome this interference. A much better procedure is to clip the chest. For several years I have included a pair of barber's clippers in my examining out-fit. It has greatly facilitated my work and added to the ease and accuracy of the examination.

Method of Breathing During Auscultation.—The manner in which the patient breathes during auscultation is of great importance. Inasmuch as the examiner should listen during both inspiration and expiration, it is well to have the patient breathe a little deeper and a little faster than normal. Forced respiration, however, should not be employed at first, for this disturbs the rhythm of the respiratory note. Deep breathing causes a comparative prolongation of the expiratory note. This prolonged expiratory note is often not heard because the examiner fails to listen sufficiently long and sufficiently intently. The breath sounds should be freed from adventitious sounds produced in the nose or larynx. Sometimes it is necessary to have the patient breathe with open mouth to overcome the sounds caused by a nasal obstruction.

The patient should be examined while breathing quietly as mentioned above, then during deep inspiration, and finally after a short cough followed by inspiration. This is particularly necessary in examining for râles. Often a deep inspiration will show râles where a shallow inspiration will not; and a cough followed by inspiration will show them, when both shallow and deep inspiration fail. No physical examination for tuberculosis and particularly for early clinical tuberculosis should be considered complete without auscultating during deep breathing, and cough at the end of expiration, followed by deep inspiration.

Abdominal breathers are often difficult to examine because of the weakness of the respiratory note 'over the apices. This can be partially overcome by instructing them how to breathe with the upper portion of the chest. In this connection it should be emphasized that the ventilation of the lungs is increased when the patient sits or stands and decreased when he lies down, consequently sitting or standing brings out the changes on auscultation more plainly.

The Effect of Muscles on the Respiratory Note.—Contrary to the usual advice about the position of the arms during auscultation, I prefer always to listen through a muscle which is relaxed as much as possible. There is a difference in tone when heard through a relaxed muscle and through one in a state of contraction. This can be readily proved by examining through the trapezius when the shoulder is raised, relaxing it, and when the muscle is taut as it is when the hand is placed on the opposite shoulder; or by listening through the pectoralis when the arm is thrown forward relaxing and then thrown backward with the arm near the side putting it on the stretch. Through the relaxed muscle the tone is soft and breezy, while through the contracted muscle it is harsher, sometimes rougher than normal, and of higher pitch. To appreciate the effect of different thicknesses of muscle on the auscultatory note, let the examiner listen in the axilla and compare it with the note over the lower folds of the pectoralis immediately above.

It is very important to appreciate this muscle element in the respiratory sound. While the sound heard on auscultation is primarily a respiratory affair, it is modified by the pulmonary tissue, the bony cage and the soft parts. Muscle movements of themselves emit quite a note. This can be easily determined by listening through the stethoscope to the biceps while the muscle is at work. It will also be noted that the difference in the note heard on easy contraction and on forced contraction is marked, the latter being not unlike the changes noted in the respiratory note in the presence of slight pathological conditions in the pulmonary parenchyma.

There is no doubt that the reflex increased tone (spasm) of the apical muscles is an element in the altered sounds that we hear in early tuberculosis the same as it is in the altered percussion and palpatory findings. How much of an element it is cannot be accurately stated, but that it is important cannot be doubted.

Respiratory Sounds in Early Tuberculosis.—There is no definite change in the respiratory note which is always present in early tuberculosis. If there was it would be a comparatively easy matter to make early diagnoses on stethoscopic findings. There is also a confusion of terms, one man calling a given sound harsh, another calling it rough. Part of this confusion is due to the changes which sound undergoes in passing through different stethoscopes, and part of it to the different methods of breathing during auscultation. The degree of roughness and harshness is often accentuated by deep breathing. It is important to remember that there are several changes in the auscultatory note in early tuberculosis, one or more of which may be present. A very common condition is what is called roughened breathing. I call this condition when very marked "near râles" because the sensation present suggests that the sound is about to burst into râles. Others characterize it as a condition of stickiness. The note is sometimes slightly harsh with higher pitch than normal. The expiration may be prolonged. An important condition is that of weakened breathing or "impeded breathing," as I prefer to call it. The breathing is not full and free. Air enters as though against an impediment, and the sounds are not as full and loud as normal. Râles may or may not be present. In truly early clinical tuberculosis they will usually be absent; and if present it will be necessary to listen to a deep breath following coughing in order to detect them.

Weakened and Roughened Breathing.—This type of breathing is often found; in fact is an almost constant accompaniment of early infiltrations of the apex. Brecke[4] quotes Strümpell, Turban, Knopf, A. Fränkel, Rumpf, and Bandelier and Roepke as agreeing that the earliest alterations in the auscultatory note in apical tuberculosis is a change to weak and rough. Many different causes have been suggested for rough breathing; one, the interference with the air entering the acini; another, secretion in the air passages; another, an admixture of sounds produced by secretion and the normal pulmonary sound; and still another suggested by Waller, the sound produced by the contracting respiratory muscles. Breeke then states that the weakened breathing has not been explained.

Another very common condition found in early tuberculosis is a diminished respiratory murmur. It seems perfectly evident to me that the lung condition, as well as the action of the muscles is a factor in its production. While it is probable that the lessened elasticity of the lung tissue is a factor, the lessened inspiratory excursion of the side which is at least partly a reflex phenomenon is equally important and the effect of the altered tone of the muscles upon the transmission of the sound must be considered. At times an adherent thickened pleura becomes an additional factor. In considering the muscle element, not only the action of the apical muscles but that of the diaphragm as well must be kept in mind. The fibers of all of these are thrown into a state of increased tone (spasm) which limits the free motion of the entire side of the chest, thus reducing the force of the respiratory murmur. That portion of the lung which is the seat of infiltration offers extra obstacles to the ingress of air as compared with the uninfiltrated lung; consequently it may show a marked degree of weakening of the note.

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[4] Brauer, Schroder, und Blumenfeld: Handbuch der Tuberkulose, Bd. i, 1914.

Quoting from a previous paper:[5]

"I have described this complex picture presented to the ear in early tuberculosis as 'impeded breathing;' meaning by this that the air enters the lung as though it were forcing its way against obstacles. The murmur instead of being smooth and of its usual volume seems to be diminished, somewhat rougher, and harsher than normal. I am now convinced that much of the change is caused by the contracted muscles rather than the air in the lung.

"It will be noted also that the auscultatory sound, as well as the data obtained on percussion, are different near the sternum and in the middle of the first interspace between the deltoid and pectoral muscles from that found over the muscle masses.

"I do not wish to be understood as claiming that all the changes which we find on auscultation are due to the muscle condition, but I wish to call attention to the fact that there is a muscular element in all auscultatory findings in the chest and that the auscultatory sound in early tuberculous infections is especially influenced by the underlying contracting muscles. After severe changes such as dense infiltration, which lead to the formation of scar tissue and cavity formation in tuberculosis, and such as occur when other pulmonary diseases are present in the lung, the muscular element probably becomes less of a factor and the pulmonary tissue more of a factor in the production of the auscultatory sounds.

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[5] Pottenger: The Importance of the Neck and Chest Muscles in the Production of the Phenomena Obtained by Percussion and Auscultation of the Chest, Archives of Diagnosis, October, 1910.

"The muscle element has been recognized in examination of the chest unknowingly; for example, teachers of physical diagnosis, and authors of text-books, often tell us that if we wish to fix in our minds the normal vesicular respiratory murmur, it is best to listen in the axilla. The reason for this is not that the sound heard there is any purer than over any other portion of the chest, but that the sound is less affected by muscles, because there are no fleshy muscles covering the chest in the axilla."

The part that the muscles play in the production of the sound heard on auscultation may be realized by comparing the auscultatory sounds heard over a rigid thorax or over the thorax of one who practices abdominal breathing with one who uses the thoracic muscles in the normal manner. The lungs are inflated in the former condition the same as in the latter. The pulmonary tissue is distended, but the sounds are weak, at times almost inaudible, and much smoother than when the muscles exert their influence more strongly upon the sounds.

Not only do the chest muscles influence the respiratory sounds as they are conducted through them to the ear, but they also produce sounds which are difficult at times to distinguish from intrapulmonary râles. One can satisfy himself on this point very readily by listening over a chest in which the pulmonary tissue is collapsed as a result of pneumothorax or empyema. At times, sounds which are difficult to distinguish from intrapulmonary râles, are heard over the apices of the lungs.

Why Respiratory Sounds Differ in Early Tuberculosis.—A discussion of why the auscultatory sounds differ in different patients suffering from early tuberculosis is of great importance in elucidating this most difficult yet most important procedure in physical diagnosis.

Sounds may vary in different patients according to the conditions which produce them and the condition of the tissues which transmit them. The voice itself differs materially in different individuals. The character of the breathing is a variable factor.

The amount of soft tissues, subcutaneous and muscular, differs greatly. The character of these tissues, whether flabby or firm, and particularly the state of the muscles, whether they are in contraction, relaxation, or whether they show degeneration is important. The state of the bony thorax whether elastic or fixed makes some difference also.

Another important factor in the variability of the auscultatory note is the pathological conditions, other than of tuberculous origin, affecting the lung itself. The lung is often in an abnormal state. It may be emphysematous; it may be the seat of fibrosis resulting from previous influenza, pneumonia or tuberculosis; it may be altered because of an old pleurisy or empyema or it may be modified by the fact that we have a new activity in an old focus. All of these factors tend to make a difference in the note to be heard even in what might be considered as early clinical signs. If we were always listening to a new infiltration in a lung which had been normal hitherto, the matter would be comparatively simple.

To my mind the harsh and high pitched characteristics are fundamental and more prominent than the rough element when we have a lighting up of activity in an old focus providing it is sufficiently extensive to result in fibrosis of considerable amount. Here the prolonged expiration is also most marked. This is also marked, however, if the new infiltration is extensive. The purest rough breathing, on the other hand, comes in the apex involved for the first time. Impeded breathing comes under both conditions but shows best in the newly involved.

Interpretation of Auscultatory Findings.—Findings on auscultation must be interpreted, as a rule, in connection with the clinical history. Now and then positive incontrovertible evidence of early clinical pulmonary tuberculosis will be found on auscultation without any symptoms being present which the patient has recognized; but this is rare. As a rule, symptoms will be present under these conditions. Râles localized at one apex are very significant; in fact, the most significant of auscultatory signs; but, they are present in only a small proportion of early tuberculous cases and should not be expected. They come after the diffusion of toxins has produced a collateral inflammation with exudation in the tissues. Rough breathing, or rough harsh breathing, or localized impeded breathing are very suspicious, and if accompanied by clinical symptoms warrant a positive diagnosis. When these are significant of an active tuberculous process, some degree of diminished motion will nearly always be present if looked for in a painstaking way; an increased tone (spasm) of some of the apical muscles will usually be found; and, a prompt maximum tuberculin reaction will usually occur.

Whispered Voice.—An intense whispered voice is of some value. This presupposes a pathological area of some magnitude, however. To determine this a syllable should be chosen which can be drawn out. "Whis-per-r-r-r," with emphasis on the last syllable is splendid, or the words, "one, two, three" may be used. Sewall[6] recommends that the stethoscope be pressed firmly to the surface of the chest in order to eliminate the muscle elements of the sound as much as possible; and that the words "one, two, three" be spoken staccato. If infiltration is present, in spite of the staccato whisper the tones will be fused. This method seems to have considerable value and when understood is not difficult to put into practice.

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[6] Transactions American Climatological Association, 1913.

CHAPTER XVI.

THE SIGNS AND SYMPTOMS OF ADVANCED PULMONARY TUBERCULOSIS.

General Considerations.—Advanced pulmonary tuberculosis is an extension from the primary metastasis in the lung. The disease is more widely spread and its pathology more varied. It shows all of the degenerative changes, as well as the proliferative changes to which tubercles are subject. Its symptomatology differs from that of early tuberculosis not only in that the symptoms are more pronounced, but because new symptoms are added as the pathological changes become more advanced. From the prognostic standpoint, in comparison with the early lesion, it is much more serious. It seems best, therefore, for the physician to bear in mind two distinct pictures,—one that of early tuberculosis, the other that of late tuberculosis; consequently, in my description I have treated them separately, although such a course calls for what might be considered unnecessary repetition.

Early tuberculosis is a localized infection in the lung which produces clinical symptoms usually of a mild character; and is attended by a favorable prognosis. Advanced tuberculosis consists of an infection in the lung which shows a marked tendency to spread and which produces symptoms of a varied nature involving every important organ of the body. The results of advanced tuberculosis are serious and permanent; and, while a clinical arrestment can often be obtained, yet it is impossible to restore the tissue which has been involved in the inflammation.

The clinical symptoms of advanced tuberculosis are those which attend a more or less extensive inflammatory process, a variable degree of toxemia, and a process which is accompanied by more or less displacement of or destruction of tissue. They differ greatly in their severity according to the pathological changes which predominate. If the disease is of the fibroid type, in which new tissue formation predominates, the symptoms are less severe than where the exudative and ulcerative type predominates. Fibroid tuberculosis, however, reaches a point at times when the symptoms which arise, as a result of the pathological changes, are more important than those produced by the disease process itself.

Variability of symptoms is due partly to the individuality of the patient. As described elsewhere in these pages, all people are not constructed after the same pattern. There are differences in body form, in the stability of nerve and psychical centers, and in the stability of the function of organs. Consequently, the nerve action which produces disturbed function or symptoms is produced by weaker stimuli in some patients than in others.

The importance of careful diagnosis in advanced tuberculosis cannot be overestimated. It is not sufficient to know that tuberculosis is present. For this a physician is rarely necessary when the advanced disease is present, but the prognosis and proper application of therapeutic measures requires a careful estimate of the condition within the chest and a proper estimate of the working capacity of the other important systems and organs; consequently, the more careful our examination and the more accurate our diagnosis, the greater our ability to intelligently treat the patient.

The family and personal history in advanced tuberculosis is practically the same as that in early tuberculosis. The reader may refer to Chapter XIV for a discussion of this subject.

It is very important to know that tuberculosis is a disease which runs an uneven course. It is marked by alternating periods of activity and quiescence. The disease often extends over a period of several years, during which time, from the small beginning at one apex, or at the hilus, there is a succession of extensions until the entire pulmonary parenchyma is involved. Advancements of the disease, as well as increases in its activity, are accompanied by an exacerbation of symptoms; and each period of quiescence is accompanied by a remission of symptoms. In case the disease is not checked, these periods of extension and activity are increased until finally the entire pulmonary area is more or less involved and the patient's life is extinguished.

Classification of Symptoms of Advanced Tuberculosis.—The symptoms of advanced tuberculosis may be classed under four heads, according to their etiology, as follows:

GROUP I.

TOXEMIA.

- Headache
- Malaise
- Lack of endurance
- Loss of strength
- Nervous instability
- Insomnia
- Loss of appetite
- Digestive disturbances of the hypotype
- Loss of weight
- Circulatory disturbances
- Night sweats
- Temperature
- Blood changes

GROUP II.

REFLEX ORIGIN.

- Hoarseness
- Tickling in larynx
- Cough
- Circulatory disturbances
- Digestive disturbances of the hypertype
- Loss of weight
- Anorexia
- Vomiting
- Chest and shoulder pains
- Hectic flush
- Apparent anemia

GROUP III.

TUBERCULOUS PROCESS *per se*.

- Frequent and protracted colds
- Spitting of blood
- Pleurisy
- Sputum

GROUP IV.

AS RESULTS OF TUBERCULOSIS.

- Respiratory changes
- Dyspnea
- Circulatory changes
- Cyanosis
- Changes on part of nervous system
- Changes in internal secretions
- Changes in blood
- General metabolic changes
- Degenerative changes
- Menstrual irregularities

The symptoms designated as belonging to the toxic group play an important part in the clinical history of advanced active tuberculosis. They are prominent in nearly every case as long as active necrosis is present. They are comparatively slight in fibroid tuberculosis and may be very severe in acute ulcerative tuberculosis, and moderately severe in the combined types. Their severity depends on the degree of activity. They are accentuated by exertion and mental depression. It is difficult to specify which group of symptoms is most important in advanced tuberculosis, for we are not dealing with them from the standpoint of diagnosis, as much as from the standpoint of their effect on prognosis. The importance of the individual symptoms varies from time to time, now one predominating and now another. The toxic symptoms, however, being an expression of central irritation and endocrine imbalance, plus peripheral sympathetic action, are extremely deleterious. Those symptoms on the part of the various organs which result from the disease itself, finally assume greater importance than they do when accompanying the earlier stages of the disease.

In Chapter XIV dealing with early symptoms I endeavored to show how the three etiological factors entered into the causation of each symptom. In the present discussion, however, I shall discuss each symptom as a whole, but would refer my readers to the previous discussion for greater detail in etiology. Oftentimes in advanced tuberculosis those symptoms which result from the disease are more serious than those actively caused by it.

Malaise, Lack of Endurance, Loss of Strength.—These symptoms show themselves early in the disease with the beginning of activity, and continue, with periods of relief intervening, as a very important part of the clinical history until the end of the disease. The seriousness of these symptoms may be appreciated by knowing that they are primarily due to the effect of harmful stimulation of the nerve centers in the central nervous system. They are exaggerated at the time of each exacerbation, and disappear partly or wholly during periods of quiescence, particularly if the patient is kept quiet. Sometimes loss of strength is so marked that even the slightest exertion seems to require more effort than the patient can make. Mental tiring is no less important and no less prominent than physical tiring. Symptoms on the part of the nervous system are varied and of great interest. Toxins from the tubercle bacilli, as well as toxic substances which are produced in the necrotic tissue, likewise those which are stored up in the body as a result of the tuberculous process all act centrally, then express their effect particularly upon the sympathetic nervous system and endocrine glands. Neurasthenia, which is an accompaniment of early tuberculosis, often becomes extremely marked in some of the advanced cases. Psychoses are also at times present. The exaggerated emotional states as mentioned in Chapter VI are here greatly intensified. For a further description of the symptoms which arise on the part of the nervous system I would refer to Chapters VI, VII and VIII.

Digestive Disturbances.—These symptoms in advanced tuberculosis are varied, and very important from the standpoint of nutrition. That they should be pronounced at times is self-evident. Patients suffering from advanced tuberculosis are victims of a toxemia not only due to the tubercle bacilli, but resulting from necrotic tissue as well, which produce a marked inhibiting action on all the vital functions, greatly interfering with the appetite, with digestion and assimilation. Reflex irritation of the vagus is also present. The symptoms may vary from a slight anorexia to a complete disgust for food, or even vomiting. The degree of severity depends quite often upon the amount of activity present in the lung, although not wholly so. We at times see patients go through periods of high fever and marked destructive changes in the pulmonary tissue with little or no disturbance in appetite. On the other hand, it is extremely common, in fact the rule, to find a partial or general inhibition of action on the part of the digestive system every time an acute inflammation in the pulmonary tissue takes place. Not only the toxemia, but the reflex disturbances in the equilibrium of the organs, which gradually results from a generally decreased nerve tone, as the disease advances in severity, is an important factor for consideration. For a further discussion of this interesting question see Chapter X.

The damming back of the blood in the splanchnic areas, as described in Chapter XI, has a marked effect in the production of symptoms. The digestion also suffers from the general wasting not only of the muscles but of the cells in general. The enforced quietude of the patient also has a deleterious effect upon the body as well as the digestion. All of the common forms of stomach and intestinal disturbances are found, such as hyperchlorhydria, hypochlorhydria, hypermotility, atrophy with dilatation, stasis, displacement, and constipation, and it is characteristic of all of them that they are more stubborn and more difficult to treat than they would be in individuals who are not suffering from toxemia, whose vegetative nervous equilibrium is less disturbed, and whose tissue tone is on a higher plane, and who do not suffer from continuous splanchnic congestion. During the acuteness which accompanies cavity formation, anorexia and vomiting are not uncommon. They occur with cough or any disturbance of nerve balance, showing that they have, at least partially, a reflex origin. Hyperstimulation or hyperirritability of the vagus is the probable cause. Nausea and vomiting may be a part of a chain of symptoms which are due to definite gastrointestinal disease. Acidosis is also an important factor in these cases. Vomiting, on the other hand, often comes on reflexly with cough. Sometimes it seems to be associated with a reflex irritability of the pharynx, and when mucus collects on the pharyngeal wall vomiting ensues. This is especially prone to occur in the early morning when the patient is expelling the mucus which has collected over night, particularly if it is tenacious.

Structural changes in the intestinal tract are not necessarily present, even when the symptoms are pronounced. The tongue shows the same changes as in the non-tuberculous. There is often a slight coating of fur, at least, in nearly all cases of advanced active tuberculosis. During the periods of acute activity, with extension of the disease; or, during the time when acute caseation is taking place, sympathetic inhibition is present, as a rule, and the gastrointestinal symptoms become more acute. The coating on the tongue thickens, the entire organ may become dry, appetite lessens and the digestive capabilities decrease. The added toxemia is probably the chief element in the production of the greater disturbances. The central stimulating effect on the sympathetics caused by the toxins and depressive emotional states when added to the peripheral stimulation which takes place as a result of increased adrenin secretion is sufficient to overbalance the vagus tonus and produce the effects mentioned.

Other symptoms on the part of the gastrointestinal tract are described more fully in Chapter X.

Loss of Weight.—Loss of weight at times becomes a very important symptom in advanced tuberculosis. It is not uncommon in the far advanced disease to see a loss of one-third of the body weight, and this is often exceeded prior to death. A loss of weight of itself is not so serious a matter. All patients will lose some at times, particularly if they are not properly conserving their strength. During the period when the disease is active, even though it is not accompanied by fever, overexertion is prone to turn the balance against the patient and cause loss of weight. A loss is also to be expected during the period when the patient is having fever.

It also comes at times when the patient is worried or anxious or pessimistic. A happy frame of mind under certain conditions of the disease is essential to the maintenance of proper body weight. The inhibiting influence of discontent and discouragement upon the gastrointestinal canal at times is the greatest factor in preventing proper gain in weight.

After patients have been ill for a long time and have developed large amounts of fibrosis in the lung, with its consequent reduced pulmonary area, and its accompanying heart strain, they are apt to assume a weight several pounds below their normal and be unable to increase it to any great extent even though the disease is quiescent. I take this to be physiological or compensatory; and, look upon the new weight level, although it may be several pounds below the normal, as being best for the patient under the new conditions of respiratory and circulatory limitations. The patient cannot maintain as much flesh under the new conditions as under the old; yet seems to be in a good state of health with the low level. Often such patients will, after a time, when the heart has strengthened and the general cellular activities have improved, put on weight without any change in the character or any increase in the amounts of food taken. When this takes place, I look upon it as a good indication.

While gaining in weight is often desirable and while the physician prefers to see his patients up to their normal standard, it can readily be understood that there are conditions under which this cannot be, except to the detriment of the patient. There are many times in the course of tuberculosis when putting on weight or maintaining the body weight can only be done at the expense of the patient, and the cost is too great for the supposed gain. The gain is psychical, the danger done is material.

If one could only put the patient's mind and the minds of his friends in the right attitude on this important question, much anxiety and fretting could be avoided, and the fight for health would be an easier one. But the impossible is expected. The gain in weight is taken as the index to the patient's improvement, because it is perceptible. He is expected to gain no matter what pathological changes are going on in his lungs and no matter what the state of his general nerve and cell tone. Not only is a gain expected to take place, but no loss of weight is expected under any circumstances. Many men continue the practice of forced feeding when they know it is wrong, simply because of the patient's unreasonable demand for a gain in weight.

Circulatory Disturbances.—The symptoms on the part of the circulatory system, which come on in the earlier part of the disease, are of toxic and reflex origin; but, later, those which are a result of the disease, become even more important. The symptoms are slight in early cases and of all degrees of severity in the advanced ones, depending on the particular pathology present. For a more complete discussion see Chapter IX.

The importance of a good heart to the tuberculous patient cannot be overestimated, for it is the organ which must bear the brunt of the disease. It is subject to the influence of the toxemias which occur both as a result of the tuberculous process and those due to other causes. It suffers with other structures of the body in the general malnutrition and degeneration; and it bears the brunt of the disease in being compelled to keep up the circulation under the changed conditions as they exist as a result of the destroyed pulmonary tissue, the altered respiratory action, and the general nerve disturbance present.

Quoting from the author's former monograph:[1]

"In order to fully appreciate the enormous strain which is put upon the heart, we must consider, first, that chronic tuberculosis of the lungs is a chronic pneumonic process; second, that as the process advances, it destroys the blood vessels and also destroys the normal lung tissue and substitutes for it either cavities or cicatricial tissue; third, with the advance of the disease, contraction takes place in one portion of the lung, while other portions enlarge, becoming emphysematous, to establish compensation; and, fourth, the advanced disease is accompanied by more or less catarrhal inflammation of the air passages.

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[1] Pulmonary Tuberculosis, William Wood & Co., New York, 1908.

"All of these conditions tend to embarrass the right heart, thus damming back the blood in the systemic circulation as well.

"The heart often learns to accommodate itself to the enormous strain that is thrown upon it because the changes come gradually, taking months and years to effect them. On the other hand, if the same amount of resistance were opposed to the heart suddenly, by an acute process, the result could be none other than disastrous.

"The rapidity and efficiency of the heart's action, then, must always receive careful attention in giving a prognosis."

The heart beat is usually increased in frequency as long as the disease is acute, although there are exceptions to this rule. The particular characteristic of the heart in advanced active tuberculosis, however, is that it is not able to measure up to extra exertion. It may be normal in its frequency, or only slightly above when at rest, but increases greatly on extra demand. Such a heart also returns to its normal beat slowly.

When the body is called on for extra work it requires extra blood. This must be drawn from the great splanchnic reservoir; but in advanced tuberculosis, owing to the lessened inspiratory act, there is an inability to furnish this extra blood as needed, and even if the heart were normal in power it would fail to measure up to the demands made on it. Dyspnea results.

The persistently high pulse which fails to yield to proper treatment, is usually associated with conditions which call for a bad prognosis for the ultimate outcome of the patient. It either shows a condition of general stimulation through the sympathetic nervous system or a greatly increased effort to maintain the circulatory equilibrium. A pulse which drops as the temperature rises is suspicious of tuberculosis of the intestinal tract, the lowering effect coming through the depressor branches of the vagus (see page 201). This is also seen at times as a result of the reflex stimulation of the vagus from the inflammation in the lung, as shown in Figs. 31 and 32, pages 203, 204 and 205.

The blood pressure is low and the entire vascular tone decreased.

The heart in early cases is often smaller than normal. I have suggested as a cause of this, the lessening of the inspiratory act which causes less blood to be delivered to the right heart in a given time, resulting in a lessened content of blood and a physiological decrease in the size of the heart. This also causes a relatively small amount of blood to be delivered to the arteries, resulting in a relative arterial anemia with lowered blood pressure. Wasting of the tissues is also a factor in lowering the blood pressure as the disease advances.

Later in the disease, the right heart takes upon itself a compensatory hypertrophy, the organ may become larger than normal; but this may again give way later as the heart yields to the general malnutrition which is present throughout the body.

Whether the heart is larger or smaller than normal cannot be determined in a haphazard way. The postmortem measurements which are of value in determining the fact that the heart is smaller than normal in tuberculosis are those which have been made on young individuals of robust build who have died of other causes during the time that their disease was active, but before the advanced wasting stage has been reached. The clinical evidence must be based on careful measurements of the same type of cases.

The clinical measurements which prove that hypertrophy of the right ventricle occurs, are those taken during a chronic process on patients in whom a considerable extra strain has been thrown on the heart as a result of destructive lung changes. Postmortem evidence must come from individuals of the same type who die of accidental cause during this period without going on to a stage of severe wasting.

The measurements of the heart in the advanced wasted consumptive simply prove that the heart has taken part in the general process. If hypertrophy is marked the organ may be larger than normal. If not, it may be smaller, but such measurements have no bearing on the question as to whether or not tuberculosis is preceded or accompanied by a small heart.

Night Sweats.—Sweats are extremely variable in tuberculosis. They may come on early and be important in diagnosis or they may be scarcely noticeable during the entire course of the disease. Sometimes during caseation and cavity formation, sweats are so severe as to drench the bedding. When night sweats are present some degree of toxemia is usually present. They are a result of sympathetic stimulation in early tuberculosis, but we must not forget that sweating is also a part of the parasympathetic syndrome.

Sweating usually occurs when the patient sleeps. When it is severe fever is usually present. It is most marked, as a rule, when rapid destruction of tissue is taking place with absorption of the products of autolysis. Under these circumstances toxemia is severe and the diurnal variation in temperature is great. As the patient falls asleep the temperature drops, and a general relaxation of the superficial vessels takes place which is followed by perspiration.

Sweats may be relieved to some extent by improving the tone of the skin, as, for example, by removing extra clothing, and by hardening by sun, air, and water baths. Sweating, while common in tuberculosis, is not peculiar to it, but may occur in other toxemias and exhausting diseases.

Fever.—Fever is so important as a symptom of tuberculosis that I shall discuss it at length in a separate chapter (see Volume II, Chapter XXX). Fever itself is not as deleterious as is usually thought. It is, however, a tangible expression of the toxemia which results from the pathological changes going on in the lung and as such aids greatly in understanding the disease. It is an error to ascribe all the ill feelings that accompany it, to the fever itself. They are due to the same cause that produces the rise of temperature.

A study of the temperature curve in its relation to other symptoms will give one a fairly accurate picture of the underlying pathology.

Rise of temperature in tuberculosis is caused by bacillary toxins and the destruction of other tissues, and is in direct relationship to the amount of pathological change present and

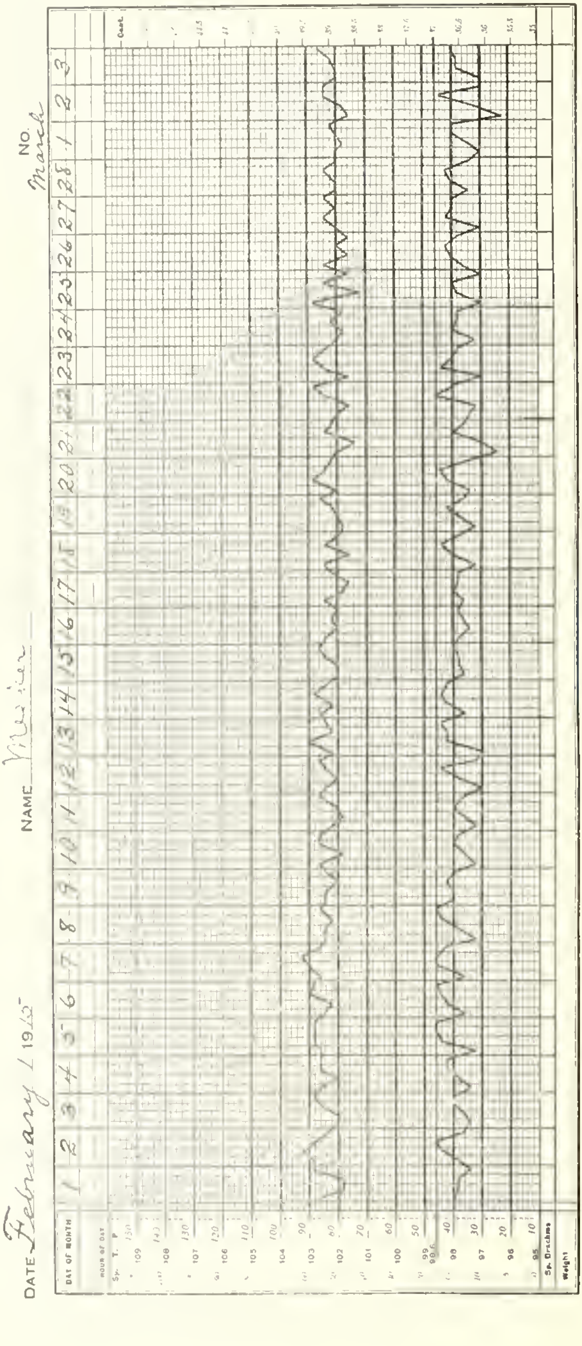

Fig. 91.—Showing a temperature curve of a patient suffering from chronic fibro-ulcerative tuberculosis during the period of quiescence. This chart cannot be differentiated from that of an early lesion with toxemia absent.

the toxins absorbed, on the one hand, and the ability of the organism to maintain its physiological heat equilibrium on the other.

Fever is not important in advanced tuberculosis in determining the presence of the disease. This can readily be determined by other symptoms and signs. The temperature curve is of great value, however, when interpreted in conjunction with the other symptoms in helping the clinician to form an opinion of the nature of the pathological changes which are taking place in the lung. It is also of great value at times in the diagnosis of complications.

The appended charts show the common curves in tuberculosis. Fig. 91 is typical of chronic fibroid or chronic fibro-ulcerative tuberculosis during the period of quiescence. Fig. 92 shows the curve of the same type of disease during periods of slight activity. Fig. 93 shows the type of temperature commonly found when the process becomes markedly active and is accompanied by extensive necrosis, and caseation and an extension to new tissue. I shall not attempt to illustrate the various types of temperature more fully at this time, but leave the subject for a more complete consideration elsewhere in these pages (see Volume II, Chapter XXX.

Hoarseness.—Hoarseness may be due to several causes in advanced tuberculosis. Probably the most frequent cause operating to produce it is reflex stimulation. This we are justified in assuming from the fact that it begins often as one of the earliest symptoms before other causes which operate later, are present, and also from the fact that it is so often exaggerated with the acute inflammatory changes which accompany the advanced disease.

The stimulation may come through either the superior or inferior laryngeal nerves. When through the former, there is a relaxed condition of the cords; they are not stretched tightly, but have a sagging appearance; the ends approximate, but the centers do not, leaving an oval slit between, as shown in Fig. 81. When through the inferior laryngeal, the cord, usually on one side, lags, failing to approximate its fellow, as shown in Fig. 82.

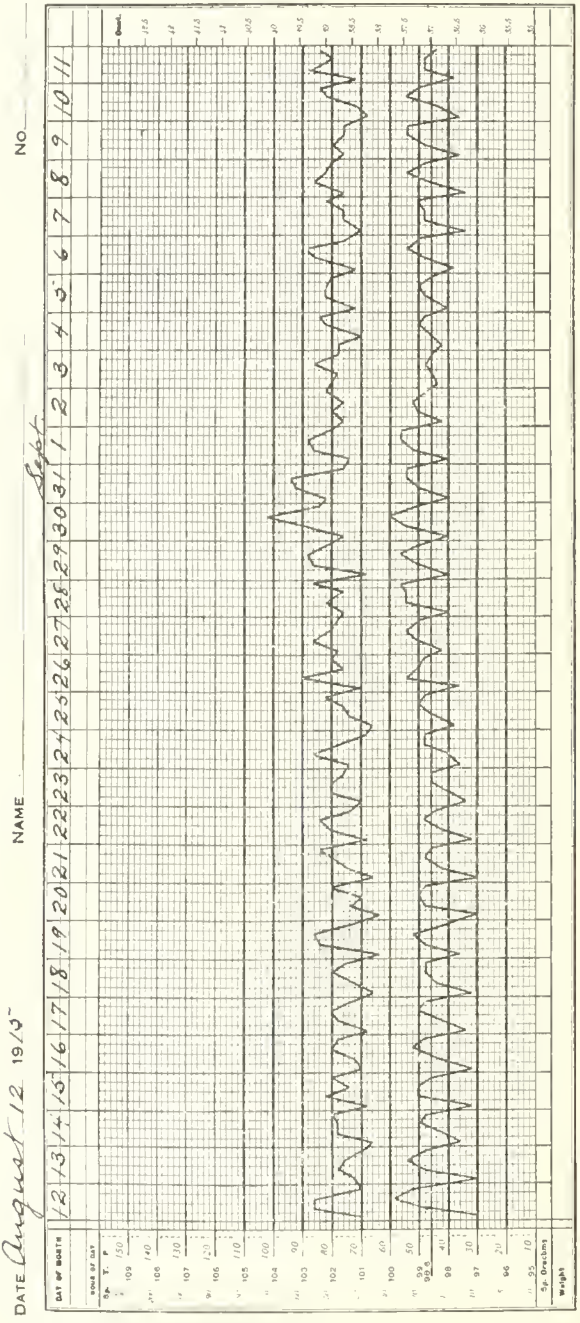

Fig. 92.—A temperature chart illustrating slight activity. From a patient suffering from chronic fibro-ulcerative tuberculosis.

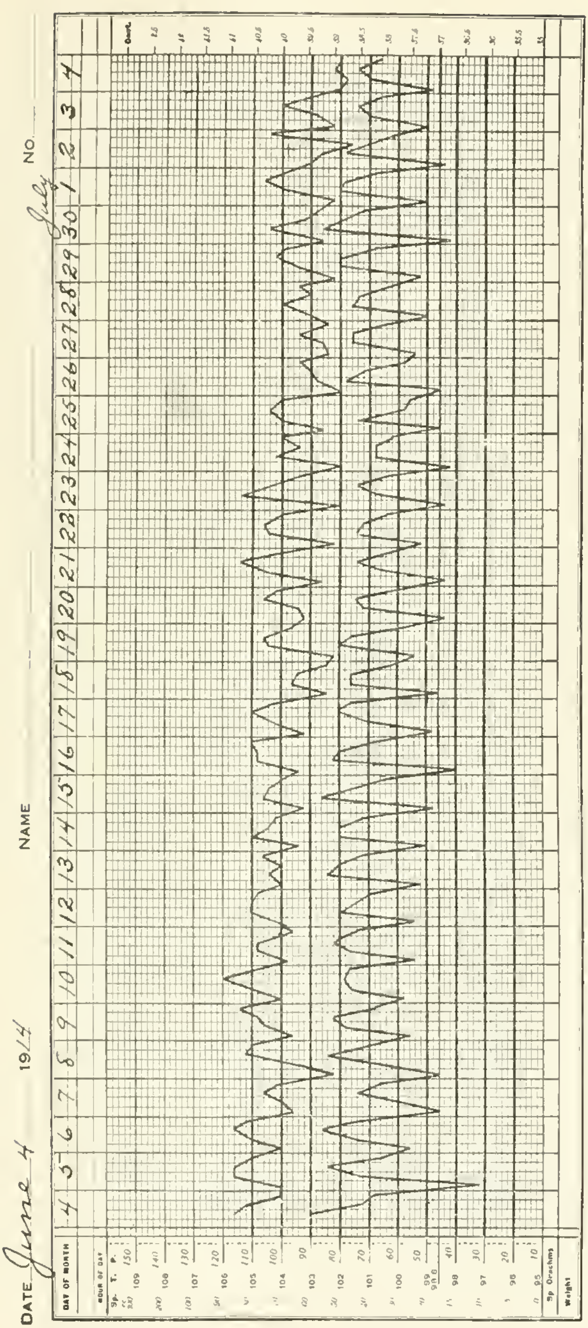

Fig. 93.—Temperature chart illustrating marked activity from a patient suffering from chronic caseous tuberculosis. The vagus influence on the pulse is noted at the end of the chart after the patient has been at rest for a period of three weeks.

While the form of nerve reflex coming through the inferior laryngeal has been recognized for a long time, I find it present as a cause of hoarseness probably less often than the type due to the superior laryngeal irritation. It is better known because we have found it in aneurisms and as a result of gland pressure. I am sure, however, that it is not necessary to have pressure on the recurrent nerve to cause it, but that it may be found as a reflex motor disturbance from the inflammation in the lung.

Hoarseness may also be due to a simple inflammation. It is important to bear in mind that, while hoarseness is often an important symptom in tuberculosis of the larynx, it does not necessarily point to it. The constant cough in advanced cases often causes an irritation of the cords which results in hoarseness. Mucus may cover them and produce it. And we must not forget the dragging on the nerves which occurs when contractions take place, resulting in shifting of the mediastinum. We often find an increased hoarseness at this time. Complete aphonia is often present as a result of reflex nerve stimulation during acute caseation. Acute or chronic laryngitis may also be present.

There is also occasionally found a reflex inflammation (neuritis) of branches of the laryngeal nerves which accompanies and results from advanced tuberculous inflammation in the lungs. It is accompanied by pain and hoarseness. It may persist for an indefinite time; sometimes only a few days, other times for weeks. The pain, and the character of the voice, is not unlike that of tuberculous laryngitis. At the time of writing this I have three such cases under observation. All have had tuberculous ulceration in the larynx which is now healed. They have enjoyed the full use of their voice for several months; and, on laryngoscopic examination, the larynx is free from active inflammation; yet, the voice is extremely hoarse, and the patients complain of pain running up into the ear, much the same as when open ulceration is present. The prognosis is good. Why this should be pronounced in some cases and not in others, cannot be satisfactorily explained at present.

Tickling in the Larynx and Cough.—Tickling in the larynx is a sensory phenomenon which has its full expression in cough.

It may be reflex, the irritation being in any part of the bronchial tubes or pleura, or it may be due to local irritation from foreign material such as mucus; or from an inflammatory process affecting the larynx itself. This irritation provokes a desire to cough. Whether the cough shall be produced or not, is largely a matter of will. A great deal of coughing in tuberculosis can be avoided if only the patient will restrain himself.

Inflammation of the pleura is nearly always accompanied by a dry, unsatisfying cough. In advanced tuberculosis, a certain amount of cough is necessary to expel the mucus, but this can be reduced to a very small amount, compared to what it would be if the patient permitted his desires to go unrestrained. When mucus is found in the bronchi it probably does not produce much irritation until it gets to the bifurcation of the trachea. This is an area of increased sensibility. Then, after passing this point, an area of decreased sensibility is again found until the larynx is reached. The bifurcation of the trachea and larynx are the two danger points of obstruction and nature has rightly endowed them with an increased sensibility so that foreign material will be quickly and involuntarily removed. When mucus reaches these points the desire to cough is increased.

The wall of cavities is comparatively insensitive. Large accumulations of secretion may remain within them without provoking cough. When they are filled, however, and overflow, and the secretion begins to gain access to the bronchi, cough is provoked; and, when the points of special sensitization just mentioned are reached, the desire is increased, and kept up until the mucus is forced on beyond and in the latter case expelled. The amount of necessary cough depends considerably upon the amount and character of secretion; and this, in turn, depends a great deal on the amount of active destruction and the degree of associated bronchial inflammation present. An acute cavity, produces more secretion as a rule, than a chronic one of the same size, consequently calls for more cough. When cavities of considerable dimension are present the patient's cough is usually exaggerated on change of posture. On awakening and moving in the morning and again on going to bed at night, a paroxysm of cough is precipitated which does not cease until the cavities are emptied; or, at least, until that portion of the secretion which gains access to the bronchi is expelled.

Changes in position which permit of the escape of secretion into the air passages cause cough. Patients learn that they can lie on one side or in one position without cough, while lying on the other side, or in another position, excites it.

While a certain amount of cough is necessary, and unavoidable, no unnecessary coughing should be allowed because of certain deleterious effects which follow it. It is an explosive expiratory act begun with the glottis closed when the pressure in the air passages is high. As a result of this there is a forcible dilatation of the pulmonary parenchyma, which has a tendency to force bacillus bearing mucus into the small bronchi, favoring secondary bronchogenic infection. It also causes a damming back of the blood into the systemic veins. Cough and deep breathing are probably the two factors which are responsible for most bronchogenic infections. Inasmuch as this is a common method by which the infection spreads, when a lung is once involved, cough and deep breathing should be avoided as much as possible. It also forcibly stretches newly formed fibrous tissue.

Chest Pains.—The classification and differentiation of pain in and about the chest is attended by great difficulty. The patient with chest pains is usually frightened and more or less certain that serious pulmonary disease is present. The symptom "pain" will cause the patient to consult a physician more quickly than any other. This being true, it is unfortunate that tuberculosis is not always accompanied by pain; but it is the duty of the physician to familiarize himself with the pains in the chest so that he may be able to assign to them their proper significance.

The pains which must be considered and differentiated in chest diseases are those due to intrathoracic organs and those due to intra-abdominal organs but which express themselves over the same or adjacent surface areas.

Pain due to inflammation of the lungs and pleura may be located over the anterior or posterior portion of the thoracic wall, in or over the shoulders, and below the costal margin.

Pain of thoracic origin may be due to diseases of the soft parts, the nerves, the bones, pleura, lungs, heart, pericardium, blood vessels, or mediastinal glands. Those with which we are particularly interested are those from the inflammation in the pleura, lungs, and mediastinal glands.

Nearly all patients experience some discomfort during the course of tuberculosis, although it is usually interrupted in character and of short duration. The reflex pains, particularly those which are due to reflex neuritis, and those of pleural origin, more particularly those due to adhesions in which the pleural and intercostal nerves become involved in the inflammation, come and go with atmospheric and meterologic changes. The same is true of the reflex pains noted in the sensory zones of the skin. When weather changes are most pronounced these pains are most annoying.

When acute cavity formation is in progress the patient may often locate the point of inflammation quite accurately by the pain experienced. Occasionally he may also suspect an approaching hemorrhage by a discomfort or pain which he has learned to associate with it. To be accurate this must be near to and involve the pleural nerve endings.

During the compensatory changes which take place when marked contraction affects one lung and a compensatory emphysema takes place in the other, adhesions are often stretched and torn with a resultant discomfort or pain. Sometimes the pain is quite acute. At times there may be no actual pain noted, yet the pullings and drawing may be sufficient to cause great discomfort, the patient complaining of a feeling of tightness or compression.

The pain resulting from coughing, particularly during the time of acute softening, when the cough is greatly increased, is felt at the attachment of the abdominal muscles along the costal arch, and is often very disturbing to the patient causing him to think a very severe inflammation is underlying it.

Our knowledge of the reflex sensory disturbances which manifest themselves as areas of hyperesthesia and hyperalgesia of the skin when the abdominal and thoracic organs are inflamed is largely due to the work of James Ross,[2] Head[3] and James Mackenzie.[4] These original contributions repay careful study.

The sympathetic nerves, which come from inflamed organs such as the lungs, when the seat of tuberculosis, or any other inflammation, carry impulses through the posterior root ganglia to the gray substance of the spinal cord and there set up in the cells a state of increased irritability. Sensory nerves which take their origin from the cells in the same segments of the cord which mediate with the sympathetics, are so influenced that their impressions are heightened. Thus, the skin supplied by them may become more sensitive than normal to such stimuli as heat, cold and touch. Painful sensations are out of proportion to their severity.

The areas of the skin which show sensory changes most in inflammation in the lung are those which are innervated by the third and fourth cervical segments. This comprises the skin of the neck and chest above the second rib anteriorly and the spine of the scapula posteriorly. The third, fourth and fifth thoracic zones in the interscapular region also show some pain, particularly in hilus infections. There may also be deep soreness in the muscles innervated by the cervical cord. These zones may be seen in Figs. 27 and 28, pages 182 and 183.

These sensory changes are not evident at all times during the course of pulmonary tuberculosis. They are most commonly found in early lesions, and at times of acute activity in the process.

Not only are there sensory changes but motor changes also, which are entirely analogous in their causation and distribution, as I have shown previously and as described in Chapter VI. Resulting from the prolonged irritation of the cells which give origin to the motor filaments they become inflamed; and this irritation, at times, shows in the nerve as a true neuritis, usually, however, of only moderate degree of severity. This is further favored by the toxemia present; but since it is so often present early, before toxemia becomes so prominent a factor, and, since it is regional, showing only on the affected side and in definite areas; I feel that we are wholly justified in considering this type of pain as due to localized neuritis caused reflexly through the sympathetic irritation of the cells which give origin to the motor filaments. Thus do we account for many of the aching pains in the shoulders, which are so commonly called rheumatism. Rheumatism of the shoulder, particularly if confined to one side and if in an individual who is below par physically, should cause the examiner to give attention to the underlying lung.

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[2] On Segmental Distribution of Sensory Disorders, Brain, 1888.

[3] Brain, vol. xvi, 1893.

[4] Sensory Symptoms Associated With Visceral Disease, Medical Chronicles, vol. xvi, 1892; and Symptoms and Their Interpretation, London, 1909.

Pains caused by pleurisy show a varied distribution (see Volume II, Chapter XXV. Not only may the pain be over the seat of inflammation, but it may extend beyond the area, and even be transferred to other nerves. Thus, diaphragmatic pleurisy may show pain and spasm over the abdominal wall through the tenth, eleventh and twelfth thoracic nerves or over the shoulder, the impulse being carried by the phrenics to the third and fourth cervical segments where the reflex is transferred to the external supraclavicular nerve derived from the third and fourth cervicals. This shoulder pain is a developmental remnant and due to the fact that in earlier life the diaphragm is on a level with these cervical segments.

The pain and spasm of the abdominal muscles, due to diaphragmatic pleurisy is, at times, mistaken for inflammation below the diaphragm, and operation has been undertaken because of it.

Intercostal neuralgia is a diagnosis often made; but like "la grippe" and "neurasthenia" the diagnosis so often means nothing. The cause of neuralgia is the important thing. Very often the diagnosis of tuberculous pleurisy would be the correct one instead.

Pain over the sternum may be caused by bronchitis, mediastinitis, aneurism, hyperchlorhydria, or disease of the sternum itself.

Pain over the right shoulder is often caused by gall bladder disease, while that over the left may be due to affections of the stomach or colon should the inflammation involve the diaphragm.

Pain over the left mammary region, radiating down the inside of the left arm is usually of cardiac origin. It accompanies myocardial degeneration and dilatation. I have also observed it in a few instances when the upper left pleura was inflamed.

In uterine and ovarian diseases pain may be found in the breast at the time of menstruation. This is commonly found in tuberculosis, but that there is a definite relation between it and the tuberculosis I have never been able to determine.

Pain in the back over the interscapular region, high up, may be due to pericarditis, disease of the hilus, diaphragmatic pleurisy and aortic lesions. Pain in the areas immediately below this is usually due to mediastinal affections. Pain near the spine, within the angle of the scapula on the left may be due to the stomach, and on the right, at the angle may be due to the liver, while pain in the scapular region is usually due to the lungs. The pain experienced in the lower part of the chest along the attachment of the diaphragm in cases of colitis or stomach affections accompanied by much gas, are often very distressing to the mind of the patient and usually considered by him to mean serious pulmonary trouble. It is necessary to bear these pains, which are over chest areas, but due to other organs, in mind for differential diagnosis.

Symptoms on the Part of the Nervous System.—The symptoms which arise on the part of the nervous system in advanced tuberculosis are many and varied in character. For their discussion see Chapters VI, VII and VIII. One cannot understand tuberculosis and the disturbed functions accompanying it without understanding the nervous system.

Acidosis.—There are probably many factors present in tuberculosis which have a tendency to increase acidosis, such as deficient intake of oxygen; deficient excretion of carbon dioxide which occurs particularly late in the disease as a result of diminished pulmonary area; slowness of the circulation owing to the peculiar vasomotor changes; also the general changes in the tissues themselves, and disturbance on the part of the heart. Toxins also act to the same end. These factors afford the explanation of the dyspnea which appears when the body is called upon for extra exertion.

This subject has received a great deal of attention recently, particularly by men who are interested in diabetes and diseases of the heart and kidney, but it also demands attention from those who are interested in pulmonary troubles.

Dyspnea.—Dyspnea is a symptom which arises in advanced tuberculosis under several different conditions. At times it comes on early in the disease. It is an indication of either a difficulty or an inability of the respiratory system to adjust itself and properly oxygenate the blood under the unnatural conditions under which it is working. From this there is an increased acidosis resulting in dyspnea as just mentioned.

Respiratory movements are for the sole purpose of keeping up the proper gaseous exchange between the body tissues and the atmosphere. Interference with this exchange may result from disturbances of the respiratory tissues themselves, such as comes in tuberculosis and other causes which interfere with the proper propulsion of the blood through the body.

When the disease is active and spreading rapidly it is impossible for the remaining portions of the lung to compensate with sufficient rapidity to avoid dyspnea even though the loss of tissue of itself is not sufficient to warrant the degree of disturbance noted. Part of the disturbance under such circumstances is undoubtedly due to the severe toxemia present, and is of nervous origin.

The most severe dyspnea that I have met in this disease was in chronic fibrosis with pleural adhesions, the process having gradually extended until it had completely used up all reserve lung tissue. Such cases, as a rule, have not only insufficient pulmonary tissue to aerate the blood, but they also have a weakened heart muscle, an altered blood supply, sometimes adhesive pericarditis, and, as a rule, pleural adhesions of such density as to greatly interfere with the respiratory act and to cause the weakened compensatory respiratory muscles to be called into operation.

Dyspnea is increased on exertion. A patient may be perfectly comfortable while resting and yet be unable to measure up to the least extra exertion without showing dyspnea.

Coughing is often sufficient to bring on such a degree of dyspnea that the patient fears for repetition of the paroxysm.

Rest is the first and most important measure for the relief of dyspnea. But measures which will benefit either the heart, the condition of the blood, or remove pathological changes from the lung, will prove helpful. Sometimes it is exaggerated by disturbances in the gastrointestinal tract, and can be relieved by attention to conditions below the diaphragm.

Not only the degree of embarrassment, but the cause of it must be taken into consideration in the prognosis.

Hectic Flush.—Hectic flush is a reflex symptom confined, for the most part, to the periods when the tuberculosis is active. It often comes on early and is found in all stages of the disease. The flush is a reflex dilatation of the blood vessels of the face, head, and ear. It is confined to the side of activity when unilateral and more marked on the side of greatest activity when bilateral (see page 199). This is both a parasympathetic and sympathetic reflex: the former through the vagus and fifth cranial to the face, the latter through the sympathetics and third spinal nerve to the ear.

Sputum.—Sputum is sometimes present very early but when marked is an accompaniment of an advanced process. There may be a slight amount of secretion present as a result of exudation or local stimulation of nerve endings even before ulceration occurs. This may be mucoid or it may be slightly purulent in character. When it is a part of the tuberculous process it may show a high lymphocyte count and have diagnostic value, even though free from bacilli (see page 536). Sputum is an important diagnostic sign for it is due to the tuberculous process *per se*. For a critical discussion of methods of examination of sputum and the interpretation of the findings, see Chapter XX, page 533.

No matter how insignificant the amount of sputum, it should be collected and examined. Much time will be saved by taking a twenty-four hour, or three-day specimen, when the amount of expectoration is slight, as it sometimes is even in advanced tuberculosis. This should be fermented and homogenized by a shaking machine or treated with antiformin before examination, as described in Chapter XX.

Sometimes patients claim that they do not expectorate, although physical examination indicates the presence of secretion. In such cases the sputum is sometimes raised into the throat and swallowed. In a few such cases we have found bacilli in the feces when we were unable to obtain a sample of sputum until we had convinced the patients of what they were doing.

Chronic fibroid tuberculosis may be extensive and yet produce little or no expectoration. The ulcerative type, however, shows varying quantities, usually large when the destructive process is acute and extensive.

Not all secretions expelled by the tuberculous patient are from the ulcers in the lung. Much of it is of bronchial origin, while some patients discharge large quantities from the throat and pharynx. Some patients with an acute destructive process in the larynx are greatly annoyed by profuse secretion.

Not only does the amount of secretion vary according to the involvement in the lung and the amount of bronchial, laryngeal, and pharyngeal irritation present, but it varies greatly according to the weather changes as is easily explained. When the air leaves the lungs it comes out saturated with moisture; so if a dry air is inhaled it will carry off far more secretion than a moist air and consequently leave less to be expectorated. In this way dry atmospheres of favored regions help to reduce the amount of secretion expectorated.

Whether the secretion is coming from cavities or from bronchial inflammation may be determined somewhat by its relationship to posture. If it is from a cavity it may accumulate as long as the position is such that the opening does not offer drainage; but, when the cavity is filled or when the posture is changed so as to favor drainage, the contents are usually emptied. Under these conditions, the patient, as a rule, has a more or less severe coughing spell on arising and again on retiring. Patients learn that there are certain postures which favor retention and assume these during sleep, otherwise they are obliged to cough during the night. Bronchial secretion, or secretion from small cavities, or from larger ones where the opening favors drainage, is apt to be expelled as it forms, during the day.

The origin of sputum cannot be told by its physical characteristics. The appearance of cavity sputum may be the same as that of bronchial origin; so may its consistency be the same. Sometimes, however, the typical nummular sputa are present, indicating that they come from cavities.

The amount of sputum varies greatly. It may be only a few cubic centimeters or it may be five or six ounces in twenty-four hours. I have seen as much as 750 cubic centimeters in a day. Large quantities may come from chronic non-tuberculous bronchitis; but, as a rule, if 15 or 20 cubic centimeters are expectorated daily, the largest portion of it coming in the early morning on arising, a cavity should be suspected.

Pleurisy.—Pleuritic pain is a common symptom in advanced tuberculosis. Its diagnostic value should always be recognized. There is no excuse for allowing the diagnosis to go unmade when the patient suffers from this symptom. It is not sufficiently recognized that pleural pain may have many characteristics and may be located in various parts of the thorax. Too often it is looked for only at the bases; and it is too often considered to be only of the sharp cutting variety. It may or may not be accompanied by a pleuritic rub.

The pain caused by pleurisy may be sharp, cutting, stabbing, dull and aching, or boring; in fact, it may have almost any characteristic experienced in pain.

Pleurisy may be dry or accompanied by effusion. The most common is the dry variety. Pleural adhesions are the result of inflammation of the pleura and are present to some extent in nearly every case of advanced tuberculosis. Sometimes they are extremely dense, and yet the patient is unable to give a history of when the inflammation occurred, because of a complete absence of pain. (For a more complete description see Volume II, Chapter XXV.)

Frequent and Protracted Colds.—Many advanced cases will give a history of repeated colds which end in bronchitis and a persistent cough. All such are suspicious. Most affections of this class are not simple bronchial infections, but accompany periods of activity in the tuberculous involvement in the lung and should be investigated carefully. It is not natural for man to suffer from one chest cold after another. Even though one was specially susceptible, he could hardly expect to have frequent attacks of simple bronchitis. Therefore, every case of this kind should be investigated for other symptoms of tuberculosis, for this is nearly always the real cause of the symptoms. The tuberculous nature of the affection should be especially suspected if symptoms of toxemia persist.

Hemoptysis.—Spitting of blood is most commonly due to a tuberculous process. It assumes many forms. It may be slight in amount or profuse. It may be bright red, pinkish, or dark colored. Sometimes blood is expectorated from the gums and occasionally from a discharging sinus. Such blood is usually small in amount and pinkish in character. That from the gums is thin, while that from a sinus is apt to be mixed with the thick mucus of the throat. Pinkish blood also comes from cavities in advanced tuberculosis. It can be differentiated from that from the gums by its consistency and from that from the sinuses by the fact that it is coughed up and is mixed with the usual cavity sputum which the patient is accustomed to raise.

Bright blood comes not only from tuberculous lesions, but small amounts may come from infarct, heart disease, trauma, or pneumonia. The differential diagnosis should not be difficult in advanced tuberculosis, however, for other symptoms are always present.

Profuse hemorrhages are not to be mistaken, although some men persist in assuring their patients that they are from the stomach.

Tuberculous women often expectorate blood at the time of menstruation.

As diagnostic signs, bloody expectoration should be sufficient for a diagnosis of tuberculosis in late, the same as in early tuberculosis, unless definite signs of other causes are evident. (For further description of hemoptysis see Volume II, Chapter XXXIII.)

Menstruation.—In advanced tuberculosis the menstrual function is greatly disturbed. It is accompanied by many symptoms on the part of other organs and an exaggeration of already existing symptoms. When the drain upon the patient's strength becomes marked as a result of combating the active disease, especially during the periods of caseation with its attendant loss of strength, the menstrual function often becomes irregular and not infrequently ceases. Formerly this was looked upon as a condition leading to tuberculosis. It was common report "that the patient's menstruation stopped and that threw her into consumption." This is still believed by many; although a little attention given to the clinical history will always reveal the presence of other symptoms of tuberculosis prior to the cessation of the menstrual flow. Before the flow ceases and where it does not stop entirely it often becomes scanty and of shorter duration.

Measures directed to the generative organs should not be employed to reestablish the flow. Nature stops it in order to relieve the drain. When the cause which made the cessation desirable has disappeared, and the patient has regained sufficient strength the flow reestablishes itself coincident with the general improvement. I have seen the flow reestablished after being absent a year or more.

Occasionally the flow does not reestablish, but a premature menopause comes on. I have seen this a few times in patients in the late thirties and early forties, even though a reasonable degree of health had been attained.

Aside from the disturbances in function just described as being due to the low tone of the patient, all other menstrual disorders which affect women are encountered and must be dealt with.

The menstrual period seems to be the time when women are less resistant to disease. That it is a time of great nervous tension is well known. The nervous explosions which precede and accompany it should be anticipated. The physician who observes his patients closely will often be able to relieve a high nervous tension, remove fears and depression, cut short what would otherwise be a spell of distrust or lack of confidence, or even anger or rage, by explaining to the patient that it is only the nervous tension preceding the menstrual period. Quiet patients often become hard to please, quarrelsome and faultfinding at this time. The whole world seems to be working against them. They feel unhappy and entertain great fears of their recovery and often become extremely pessimistic as to the cure of tuberculosis in general. Difficulty of sleeping is common. This is the time when a word of discouragement will be magnified many fold and unless counteracted, depression may lead the patient to give up hope. The physician must anticipate these nervous manifestations and explain them to the patient. It might be thought that once telling is sufficient but that is not so. It is forgotten as soon as the symptoms pass away and it will be necessary to repeat the same assurances the next time, and the next. Gentle reason will nearly always be appreciated by the patient.

An exaggeration of symptoms is also common at this time. The normal premenstrual disturbance in temperature sometimes assumes an acute type, a rise of one or several degrees taking place; tuberculous masses are apt to caseate, and cough and expectoration increase. If the patient is suffering from diarrhea it is apt to be worse; if constipation is present it is often more obstinate. For a more complete discussion of the physiological characteristics of the menstrual cycle, see page 195.

CHAPTER XVII.

THE PHYSICAL EXAMINATION OF THE PATIENT IN ADVANCED PULMONARY TUBERCULOSIS.

The general rules for the examination of the patient, as laid down in connection with early tuberculosis in Chapter XV should be followed.

INSPECTION.

Recalling our general remarks, inspection can be carried out much more efficaciously when the patient is sitting than when lying. This is not only because of better distribution of light, but because the respiratory act can be better studied. It is easier to study the respiratory movements of the lower portions of the chest when the patient is seated higher than the examiner, and as this is valuable in determining the diaphragm reflex in early tuberculosis and the compensatory changes in advanced tuberculosis, I prefer such a position.

Ideal conditions for inspection are a warm room, a revolving stool for each the patient and examiner; the patient stripped to the waist; the waist band loosened; and a strong light, preferably daylight, shining directly on the patient from the front.

Inspection alone, if sufficiently searchingly carried out, will give the examiner a fair idea of the pathological changes which have taken place within the chest and of the effect of such changes on the patient. In most cases it is possible by studying the effects of the motor and trophic reflexes on the muscles and subcutaneous tissues, the movements of the chest wall, the flattenings and bulgings of the bony thorax when these are present, and the position of the heart beat, to determine with surprising accuracy the nature and extent of the pathological changes within. Inspection reveals more in advanced tuberculosis than in early.

Inspection calls for the closest powers of observation and the keenest analysis of data. It is not only necessary to note departures from the normal, but such departures must be assigned to their cause. They must be dealt with as being expressions of definite pathological changes. The skin should be inspected, noting cyanosis, edema, enlarged vessels, atrophies or any other departure from the normal. Any enlargement of the lymphatic glands or the thyroid should be noted.

Atrophy and lower position of the breast upon the thorax is often to be seen on the affected side.

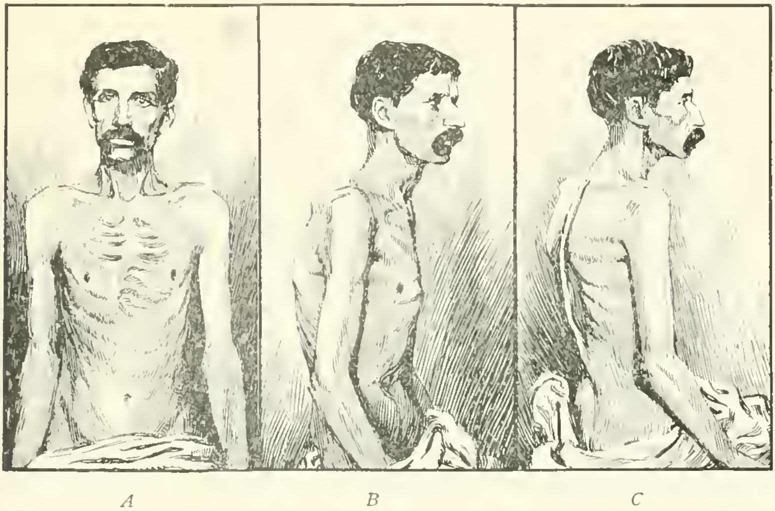


Fig. 94.—Phthisical chest. *A*, anterior view; *B*, lateral view; *C*, posterior view. (West.)

The form of the chest should be observed. Much has been written about the phthisical chest (Fig. 94, *A*, *B*, *C*) which is characterized as being long, flattened, particularly above, with acute costal angle, winged scapulæ, wide intercostal spaces, cupped supraclavicular fossæ, a drooping forward of the shoulders and a poorly developed musculature. This type of chest has been handed down through the ages as belonging to a group of individuals which is specially predisposed to tuberculosis. Some of these characteristics belong to two different conditions, the so-called *phthisicus habitus and asthenia congenita universalis. Phthisicus habitus* must be looked upon as being a result of tuberculous infection, instead of a predisposing cause, while *asthenia congenita universalis* with its long chest, wide intercostal spaces, acute costal angle and poorly developed musculature is an expression of congenital weakness. Such patients sometimes develop tuberculosis the same as those of robust build, but it is questionable whether this type of individual furnishes more than its due proportion of tuberculous infection.

Reflex Spasm and Degeneration of Muscles, Subcutaneous Tissue and Skin.—The condition of the muscles and subcutaneous tissue of the advanced tuberculous patient is important in diagnosis.

Varying degrees of spasm of the muscles exist and varying degrees of atrophy of all the soft parts, including the skin, subcutaneous tissue and muscles take place according to the underlying pathological changes.

Spasm or increased tone in the muscles of the neck, particularly the sternocleidomastoideus, scaleni, trapezius, and levator anguli scapulæ as it appears in advanced tuberculosis, is nearly always a regional reflex expression of active inflammation in the underlying lung. Where one lung, however, has undergone marked destruction and contraction, and the other has taken upon itself a high degree of emphysema, also in asthma the accessory muscles of respiration, particularly the sternocleidomastoideus and scaleni may show increased tone because of the extra work thrown upon them. Extremely rarely this may cause some confusion in that the increased tone is taken as indicating inflammation of the underlying apex. Careful analysis of all conditions present will usually make the diagnosis plain. Atrophy of the soft parts, must be looked upon as being expressive of a chronic, or it may be healed, inflammatory process in the underlying lung or pleura. It may also be due to occupational change in the muscles; and at the time of observation may be caused by a general wasting. Close observation will usually reveal the nature of the atrophy, whether regional or general.

Regional atrophy is most easily observed in the tissues of the supraclavicular notch and those between the nipple and the clavicle, anteriorly; and the tissues above the spine of the scapula running up on the neck, and those of the interscapular and sub-

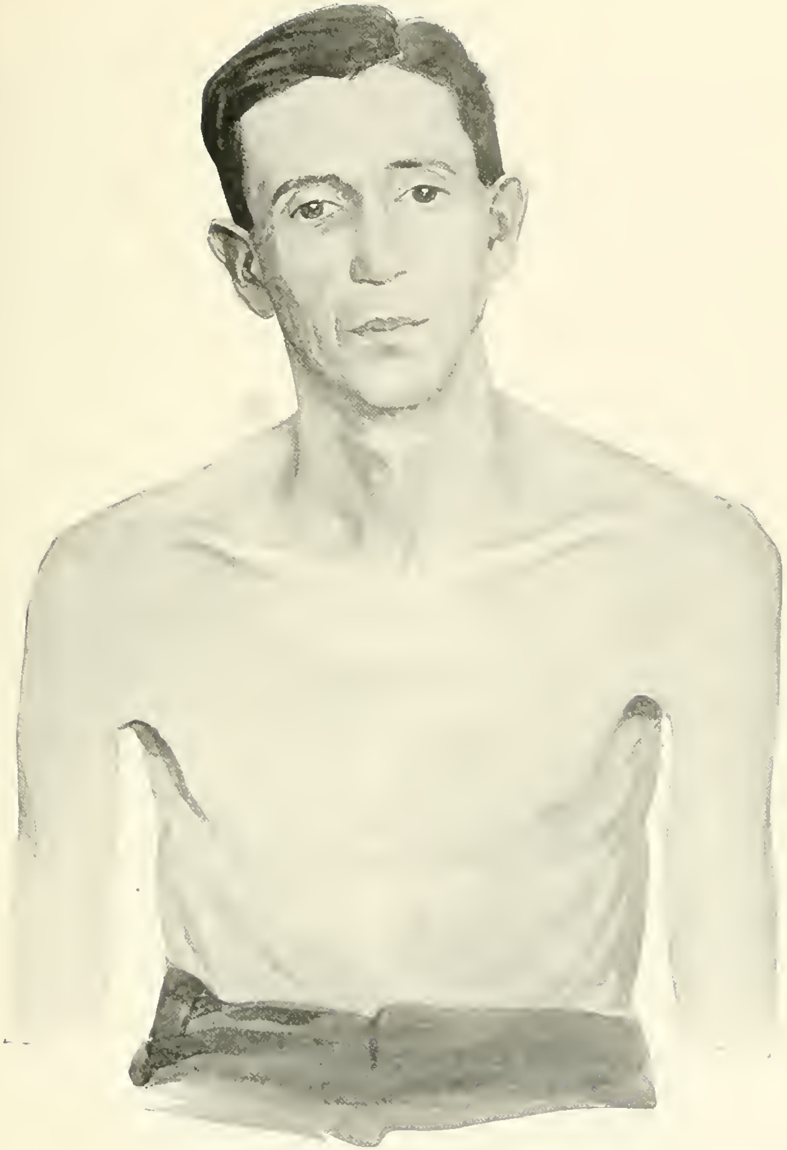


Fig. 95*A*.—Illustrating marked regional degeneration of the muscles and other soft tissues over the anterior surface of the chest as a result of chronic tuberculosis. The lesion is older and more extensive on the right side. The degeneration of the soft tissues on the right is particularly marked. The lowering of the angle of the trapezius is well shown both anteriorly and posteriorly; so is the degeneration of the right sternocleidomastoideus as compared with the left.

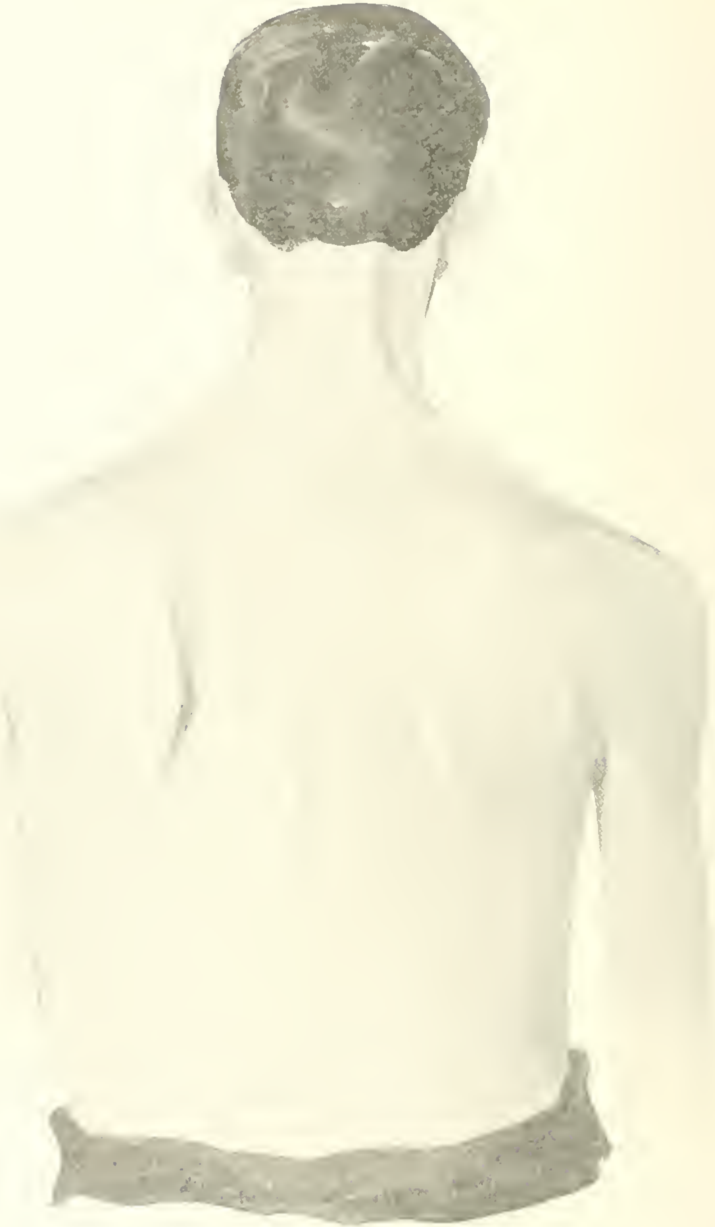


Fig. [illegible] Same as Fig. 954, showing posterior view. The right trapezius and other soft tissues are wasted more than those on the left and permit the shoulder to drop markedly.

scapular regions posteriorly (Fig. 95, *A* and *B*). While other portions of the chest's coverings show reflex atrophy at times, these are the parts most commonly affected and best studied from a diagnostic standpoint.

General atrophy, as a part of the general wasting process, does not, as a rule, affect even the advanced tuberculous patient until he has reached the acute temperature stage and has suffered considerable loss of weight. Exceptions to this are now and then found in chronic progressive fibrosis, in which a general wasting may precede ulceration. As a rule, however, only regional atrophy of soft parts will be found in the tuberculous up to the time that the loss of several pounds of weight has been sustained. Atrophy confined to one side is always suggestive of reflex degeneration.

Increased tone of muscles is not so easily observed on inspection in advanced tuberculosis as it is in early cases, but it will be observed at times. Regional atrophy, on the other hand, is observed at times. Localized atrophy of the skin and subcutaneous tissues over the neck and chest points to chronic inflammation of the underlying lung or pleura; if above the second rib anteriorly and the spine of the scapula posteriorly, it indicates a trophic reflex from the lung; if in the interscapular space, a reflex from the hilus; if below the second rib anteriorly and the spine of the scapula, a reflex from the pleura. When this difference in the trophic reflex from the lung and pleura is appreciated, it will aid diagnosis. A sign of exceptional worth is a change in contour of the neck and shoulders, which occurs as a result of the wasting of the trapezius. As the trapezius runs up into the neck to be inserted into the skull it normally forms a gentle curve; but, when it has undergone degeneration, it wastes and forms an angle more or less sharp, at a lower level than the normal curve, as described on page 407. In slight lesions this is often discernible and in widespread processes it is very marked, as shown in Figs. 95, 96, and 97. The latter two illustrations are taken from Brauer, Schröder and Blumenfeld: Handbuch der Tuberkulose, vol. i, 1914. These figures are shown by Brecke to illustrate topographical percussion of the apices. I have reproduced them to show how the degree of retraction of the apex may be inferred from inspection. The trophic changes in the muscles of the neck and upper chest suggest that the underlying lung tissue has been the seat of a chronic inflammatory process.

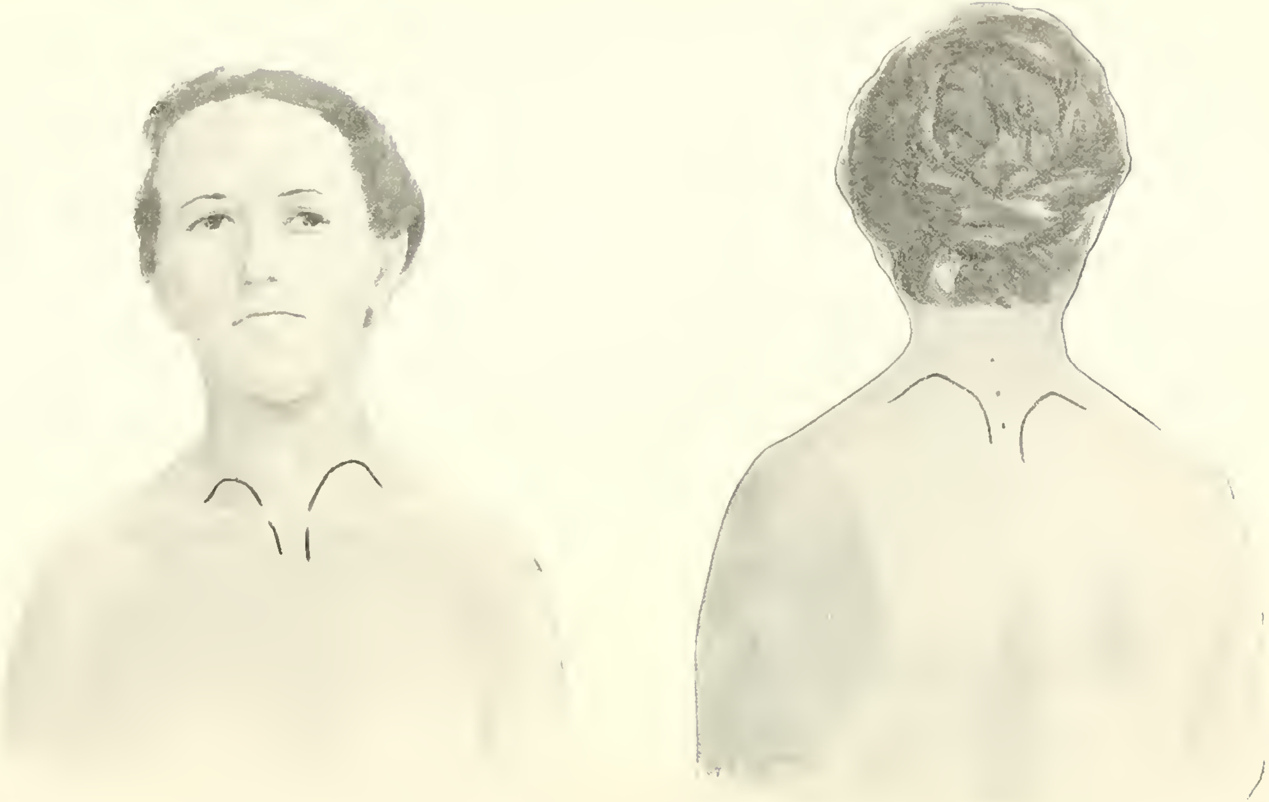
Atrophy of the skin shows as a thinning process, but it is not always easy to detect on inspection, even if it is widely distributed. Atrophy of the subcutaneous tissue, on the other hand, is usually easy to detect by the eye and wasting of the muscles is often quite plain. All of these changes are confirmed by palpation.

Changes in Contour and Movement of Chest Wall.—Next to the tissue changes above described, or of equal importance on inspection, is a careful observation and analysis of the changes in contour and movement of the chest wall. Change in either contour or movement means reflex action, changes, or compensatory changes in the tissues within, all of which (barring a few congenital conditions) occur only as a result of pathological processes; consequently, any departure from the normal form of the chest, or any variation in the normal symmetry of the respiratory movements should be looked upon as being expressions of past or present disease within the chest, and carefully studied for their diagnostic bearing.

Deficient movement of the chest wall may be noted at an apex and suggests reflex spasm of the apical respiratory muscles and reduced expansion on account of the pathological changes in the underlying pulmonary and pleural tissues. The limited motion may also affect the base, being due to a localized inflammation of the pleura; or inflammation of the lung causing reflex stimulation of the diaphragm through the phrenics and the other respiratory muscles through their respective nerves; or to a generally reduced elasticity as a result of extensive disease in the lung. This limited motion of the entire side may be seen either when the disease is limited to a small pulmonary area at the apex, or due to a general infection. There is also more or less general limitation of the motion of the entire side, even though the cause is a localized pleuritis.

It is impossible to discuss changes in contour apart from changes in movement; because if there is a flattening or bulging of the ribs it means that there must be a departure from the normal in the movements of the parts.

A



A. B.

Fig. 97.—Showing patient actively ill for six months of tuberculosis of the right apex, with some involvement in the left. Lines indicate apical outline. (Brecke.) From the degeneration of the muscles and soft tissues on the right side of this patient, barring occupational influences, one may be sure that the right apex has been involved over a prolonged period of time and that this lesion is

If a flattened appearance is noted over one or the other apices, it should be carefully examined to see if it is a real flattening of the chest wall or only a wasting of the soft tissues, which often gives the same appearance; or both. Either condition is suggestive of chronic inflammation in the lung or pleura underlying. Should the flattening be at the base, pleurisy with adhesions is suggested. Wherever the flattening is, it is usually accompanied by a limited motion of the part of the chest wall overlying it.

Enlargement and bulging of portions of the chest wall are also very commonly found. They are usually due to compensatory emphysema and indicate that a destructive process has taken place in other portions of the same lung or in the other lung, which has caused the underlying pulmonary tissue to enlarge. The respiratory movements on the side of the emphysema are usually greater than normal, while those on the other side, are less than normal.

Bulging may also be due to fluid or air in the pleura.

A correct differential diagnosis can often be made by careful analysis of the data on inspection.

A limitation of motion on one side nearly always means acute or chronic disease within the lung or pleura on that side, while increased motion means, as a rule, compensatory enlargement because of an extensive destructive process on the other side.

The following cases illustrate the relation of careful inspection to diagnosis in advanced tuberculosis:

Case I.

Mr. C. Extensive fibroid tuberculosis in right lung, with cavity at apex; compensatory emphysema at right base; thickened pleura at right base; chronic fibrosis upper portion left lung; compensatory emphysema throughout left lung; heart displaced slightly to the right; right diaphragm displaced upward; left diaphragm displaced downward.

Inspection of Muscles and Subcutaneous Tissue.—The chest is long and flattened superiorly. The neck muscles and subcutaneous tissue over them, as well as those below the clavicle on both sides, appear degenerated, this being more marked on the right than on the left. Both sternocleidomastoidei stand out more prominently than normal because of the degeneration of the subcutaneous tissue surrounding them.

From the general atrophy of the muscles and subcutaneous tissue over the entire right chest anteriorly, I would infer that there is chronic inflammation in the lung, showing above the 2nd rib, and of the pleura, showing below the same. The atrophy of the muscles and subcutaneous tissue on the left is confined to that portion of the chest above the second rib, indicating a lung lesion but normal pleura.

Inspecting the chest posteriorly we first notice that it is long, also fairly broad. We next notice that the muscles over the right shoulder, with the subcutaneous tissue over the spine of the scapula, are markedly degenerated, the right shoulder being considerably lower than the left. The occipital portion of the trapezius is more degenerated than that on the left side. The degeneration of the muscles and subcutaneous tissue runs almost to the base on the right, the latter showing both lung and pleural involvement. There is also a degeneration of the muscles and subcutaneous tissue at the upper left, though of lesser degree and lesser extent than that on the right.

Motility.—Different portions of the chest anteriorly show unequal motion. We can divide the chest into two portions, with a line running from the left acromial process to the sixth rib in the anterior axillary line on the right side. Above this line we have decreased motion and below increased motion. The increased motion is greatest on the left. From this we can infer from inspection that there has been a destructive process above this line, more severe on the right and less extensive in the upper left. We can further infer that compensation has occurred and that the lower part of the left lung is the seat of a marked and widespread emphysema, and the lower part of the right is the seat of a less extensive emphysema.

Posteriorly, the chest is not so distinctly divided as to motion, although a line drawn from the outer third of the shoulder on the left to the lower angle of the scapula on the right will divide the posterior portion of the chest into two areas of greater and lesser motion. The motion, being so much less at the upper right than at the upper left, we would infer that there has been a much greater destructive process in the upper portion of the right lung than in any other portion of the chest. Altered motion in the upper part of the left as compared with the lower portion of the left, would also indicate that there has been some destructive process in the upper portion of the left lung. Greatly increased motion throughout the left, and particularly at the base, would indicate the presence of emphysema. The increased motion at the right base as compared with the upper portion of the same lung, would also indicate that there is some emphysema at this point.

Case II.

Mr. P. Chronic fibro-ulcerative tuberculosis of left lung, with large secreting cavities in upper and lower lobe; lung very much contracted; entire mediastinum drawn to left; scattered healed tubercles throughout right lung; marked compensatory emphysema; trachea to left of median line; heart, left border anterior axillary line, right border to left of median line; left diaphragm markedly displaced upward; right diaphragm markedly displaced downward. At present time condition quiescent.

Inspection of Muscles and Subcutaneous Tissue.—Anteriorly there is a flattening over both upper lobes. The left side somewhat more depressed than the right. The motion throughout the left side is diminished—markedly so at the base. The left sternocleidomastoideus is markedly degenerated, but stands out more prominently than normal because of the degeneration of the subcutaneous tissue over it and the retraction of tissues under it. The right sternocleidomastoideus with the subcutaneous tissue over it is degenerated. The supraclavicular notch is deepened on both sides. The left pectoralis and the subcutaneous tissue over it is markedly degenerated to the fourth rib. The trachea can be seen displaced to the left. The heart pulsation is increased in an area extending from one and one-half inches to the left of the left nipple to an inch to the right of the nipple.

Posteriorly, the left shoulder is much lower than the right and the left side of the chest is smaller than the right throughout. The right is somewhat bulging. The angle formed by the junction of the middle with the occipital portion of the trapezius is one inch lower on the left than on the right, while the trapezius, levator anguli scapulæ, rhomboidei, and the subcutaneous tissue over them, extending to the base, is markedly degenerated. On the right the trapezius and levator anguli scapulæ are also slightly degenerated.

Motility.—The motion on the left is diminished throughout and on the right side is greater than normal.

From inspection alone we cannot tell whether or not activity is present; but, from the fact that the left side of the chest is markedly smaller than the right, together with the lessened motion and the atrophy of the muscles and subcutaneous tissue over this side; and the fact that the trachea runs toward the left above the jugulum; and that the maximum impulse of the heart extends to the left axilla, we infer that there has been an extensive chronic destructive process involving the left lung.

From the fact that the muscles over the apex and the subcutaneous tissue covering them on the right side are degenerated, we would infer that there has also been a chronic inflammation affecting the underlying lung on this side. From the bulging of the right side and the increased motion throughout we would infer that there is a compensatory emphysema involving the entire right lung.

The position and motion of the acromion processes (Korani); drooping of the shoulders; shape [scaphoid scapulæ (Graves)], position, and motion of the scapulæ, are all of diagnostic import. One should train his powers of observation. It is only necessary to look at chests carefully and analytically in order to form a fairly correct idea of the lung and pleural pathology within.

We do not expect to find the classical *phthisical habitus* except in a small proportion of cases. We expect to find chests of the same size and shape as we find regularly among well people, except as they are deformed as a result of the tuberculosis.

PALPATION.

When the data which may be derived from palpation is added to that which may be derived from carefully inspecting a chest, sufficient information is at hand, in the majority of cases, providing it is carefully analyzed, to determine:

1. Whether or not there is a pathological process in the lungs.
2. Its extent.
3. Whether it is active, chronic, or healed.
4. Whether sufficient destruction has occurred to produce:
    (a) Compensatory emphysema.
    (b) Compensatory shifting of the mediastinum.
    (c) Compensatory shifting of the diaphragm.
5. Whether or not pleurisy is or has been present and if so whether it has resulted in marked adhesions.
6. The nature of the process, the density of the infiltration, and whether or not destruction of tissue with cavity formation has occurred.

Inspection and palpation have not received the attention that they deserve. They require no greater skill on the part of the examiner than percussion and auscultation, but they require to be practiced; and, the data obtained requires thoughtful analysis if they are to be of great value to clinical medicine.

Palpation as usually practiced is of limited usefulness. It is confined to determining the vocal fremitus, to demonstrating pleural friction or the fremitus produced by râles, noting enlarged glands, locating the apex beat, determining expansion and eliciting pain. Determining these is the least important function of palpation. Valuable information is obtained by knowing the location of the heart beat and the altered conducting power of various pulmonary areas, together with the expansile activity of various portions of the chest. The greatest value, however, comes through:

1. Studying the conditions of the skin, subcutaneous tissue and muscles with reference to the reflex motor and trophic changes which they undergo in the presence of acute and chronic diseases within the thorax.

2. Carefully noting the departures from the normal in the

movements of the various parts of the thorax in confirmation of the alterations noted on inspection.

3. Noting the differences in density (resistance) over the various portions of the lung suggestive of infiltration, cavity, fibrosis, emphysema, thickened pleura and pleural effusions; and noting the shifting of the mediastinum and borders of the lungs.

Palpation of Muscles and Subcutaneous Tissue.—The importance of palpating the soft parts covering the bony thorax for degenerations confined to limited areas in advanced tuberculosis, cannot be emphasized too strongly, for each organ within the body is so connected through its sympathetic nerves with the spinal nerves that any inflammation in that organ reflects somewhere on the surface in sensory, motor and trophic disturbances. The point where these reflex manifestations shall show themselves is determined by the nerve filaments which take their origin from the segments of the cord which receive the impulses from the nerves coming from the organ in question. In the case of the lung, the cervical segments particularly the third and fourth, receive the impulses and these give out sensory and trophic impulses to the skin and subcutaneous tissue of portions of the neck, arms and chest, and motor and trophic impulses particularly to the muscles of respiration, including the diaphragm.

As previously mentioned, this circumscribed (regional) atrophy is not the same as the general wasting. It occurs whenever and wherever an inflammation has existed long enough to become chronic, whether it was sufficiently extensive to interfere with the general health of the individual or not. We detect localized areas of atrophy of the skin and subcutaneous tissue over the supraclavicular notch, or over the first interspace, or above the spine of the scapula, also the wasting of the sternocleidomastoideus, upper fibers of the pectoralis and trapezius when the underlying lung is the seat of a small, chronic or healed tuberculous lesion. We see this become widespread, involving the tissues over the entire chest to the costal arch and to the base of the bony cage posteriorly when the pleura becomes adherent or chronically inflamed. If the pathological process in the lung remains confined to one lung, the atrophy of the skin, subcutaneous tissues, and muscles will be confined to the same side, until such time as the general wasting is sufficient to manifest itself, then all the tissues of the body partake of it, and the regional wasting is overshadowed by the general process and is hard to determine. Prior to this time, however, any atrophy of the soft parts should be carefully scrutinized to see if it is limited in its extent, and regional in character.

The regional wasting can be determined by inspection, but it is confirmed, and at times, better determined by palpation.

When the palpating fingers press upon the soft tissues which are atrophied, several different conditions may be noted. The skin may feel thin and loose. If taken between the fingers it may be noted that the skin over the first interspace is thinner than that over the first on the other side or that over the second interspace of the same side, in case no underlying pleuritis exists.

The subcutaneous tissue appears to be wasted. The usual cushion is not there. The fibers of the underlying muscles or the underlying bones are felt more distinctly than in areas close at hand or those on the other side. The skin is not bound firmly to the underlying tissues but can be picked up and lifted from them with ease.

The muscles feel wasted. The bundles are looser and more freely movable in the tissues surrounding them than they should be—looser than those adjacent. The bundles are made out more easily and they separate more readily than they do in normal muscles. The substance of the muscle, as a whole, is reduced in amount. This shows exceptionally well in the sternocleidomastoideus where it can be taken between the thumb and finger and its size and texture compared with its mate. The degree of wasting present can also be detected by pressing the soft tissues gently against the chest wall and noting the comparative lack of tissue where the regional wasting has taken place.

A normal muscle is more or less compact, often rotund, and feels like one firm elastic mass, while a wasted muscle has lost its tone as well as compactness and rotundity.

Finally the feeling of the tissues which are atrophied is distinctive. The normal elasticity is gone and the tissues give a doughy sensation to the finger.

While spasm of the muscles may often be detected in advanced cases when the disease in the lung is a renewed activity in an old focus, yet this is attended by difficulties owing to the increased tone (spasm) being difficult to recognize on account of the atrophied muscle. Increased tone (spasm) is important, however, in determining whether the disease has extended to the other side and in determining whether it has extended to other areas in the lung.

Degeneration of the muscles covering the apex, and the skin and subcutaneous tissue above the second rib anteriorly and the spine of the scapula posteriorly, is suspicious of a chronic tuberculous lesion, affecting the underlying lung. If the degeneration extends lower than the second rib, and lower than the spine of the scapula, this is suspicious of a chronic tuberculosis of the pleura. If the skin and subcutaneous tissue over the rhomboidei are degenerated, it indicates that the hilus is the seat of an old or a chronic inflammation, usually tuberculosis.

Muscles also degenerate and lengthen as a result of use. The drop of the right shoulder in part is due to this. It is, at times, difficult to tell whether a right-sided degeneration of the apical muscles is due to occupational influences or to reflex trophic disturbances. Conditions found in these muscles are at times difficult to interpret. Two facts have helped me: first, the sternocleidomastoideus is not subject to many occupational influences; so a one-sided spasm or degeneration of the apical group of muscles, including this one, points to a reflex trophic cause for the degeneration: second, the skin and subcutaneous tissue does not atrophy as a result of occupational influences, but does so as a result of reflex trophic irritation. Again, if the atrophy is due to pathological changes in the lung, this will be detected on deep palpation, percussion, and auscultation.

Motility of Chest Wall.—Either a limitation or exaggeration of movement of any portion of the chest is at times more easily detected by palpation than by inspection. Any such departure from the normal movements of the chest is suspicious of pathological changes either in the lung or pleura, or both.

In advanced tuberculosis lessened motion of the diaphragm or chest wall may be due to acute or chronic inflammation of the pleura, or to an active infiltration or an old chronic fibrosis in the lung. Increased motion of the diaphragm, or chest wall, is a compensatory phenomenon and may be found on the side opposite to an acute pleurisy or on the side of a compensatory emphysema.

A limitation of motion of the upper portion of the chest may be due to an active inflammation at that apex, the lessened motion resulting from the combined action of the thickening of the pleura and the reflex spasm of the respiratory muscles fixing the thorax, and the lack of elasticity in the tissues, resulting from the infiltrating inflammation in the lung; or fibroid changes in the pulmonary tissue.

Departures from the normal respiratory movements can best be interpreted when considered in conjunction with the trophic and motor changes in the soft parts.

Determination of Different Degrees of Density by Palpation.—"The principle involved in palpation and percussion is the same when the interpretation of the latter is based on the sensation conveyed to the finger rather than to the sound emitted by the blow. Palpation, no matter how delicately carried out, sets up vibrations and disturbs the equilibrium of the tissues on which it is practiced. The touch starts waves which penetrate the tissues and, according to the manner in which these waves are interfered with, do the sensations conveyed to the finger differ. So, whether we generate the vibrations by touch or by a gentle stroke the effect is the same, a varied penetration and varied interference with the vibrations and a varied perception through the palpating finger, which is interpreted as meaning different degrees of density of the underlying tissues."[1]

By carefully palpating, the examiner will learn that different degrees of density are transmitted to the finger through the sense of touch, the same as they are through sound or percussion resistance. Conditions which are recognized by differences in pitch and quality of the note elicited on stroke, and by different degrees of resistance to the finger used as a pleximeter on percussion, also convey different impressions through the sense of touch by which we are able to recognize normal and abnormal conditions within the body. Light touch palpation affords the greatest acuteness in interpretation because the sense of feeling in the finger pulps is utilized to greatest advantage. Various degrees of palpation should be utilized, however, for different conditions, as the observer will soon learn for himself.

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[1] Pottenger: Muscle Spasm and Degeneration and Light Touch Palpation, C. V. Mosby Co., St. Louis, 1912.

The normal lung, the borders of the lung whether within normal limits or shifted, infiltrations, fibroid areas, cavity, emphysema, pleural effusions, pneumothorax, thickened pleura, mediastinal tumors and enlarged peribronchial glands; in fact all conditions which can be determined by percussion, can be determined by palpation. The peculiar characteristics of each of these as determined by the palpating finger I shall discuss later in conjunction with the other methods of examination.

PERCUSSION.

The principles and technic of percussion are the same as in early tuberculosis as discussed on page 417, although a greater variety of changes will be noted because of the more varied and more extensive character of the pathological changes found.

In such a complex condition as advanced tuberculosis, where all grades of infiltration, various degrees of excavation and compensation, such conditions as pleural effusion, pneumothorax, thickened pleura, and compensatory emphysema are found, it can readily be seen that we must draw conclusions from percussion only after careful consideration. Not only the changes in percussion note, but the different degrees of resistance to the finger must be carefully noted.

The condition of the muscles and subcutaneous tissue must also be carefully observed, and allowance must be made for increased tone as noted when the muscle is overdeveloped or thrown into spasm reflexly, and decreased tone, when it and the subcutaneous tissue is degenerated as a result of occupational change or reflex atrophy. Any increase in muscle substance or any increase in tone in individual muscles is followed by an impairment of the note and higher pitch, also by an increased resistance to the finger. Any wasting on the other hand, no matter from what cause, is followed by a corresponding reduction in dullness and lowering of pitch, as well as a reduction of resistance to the finger. For a more complete discussion of factors which alter percussion findings see Chapter XV.

AUSCULTATION.

Auscultation in advanced tuberculosis shows many departures from the normal in the respiratory sounds. These departures consist of a disturbance in the rhythm, pitch and quality of the respiratory murmur; a number of adventitious sounds; and alterations in the manner in which the voice is transmitted.

It is impossible to describe in detail all the changes which occur in the respiratory murmur in advanced tuberculosis for they vary under different circumstances. While there are certain characteristic sounds which belong to infiltration, others to softening and cavity formation, and still others to fibrosis; yet these processes are found in such varied combinations and differ so in their extent; and the sounds are so modified by the state of the remaining portions of the lung, the condition of the pleura and the state of the muscles and subcutaneous tissue through which they are heard, that we must be prepared to find all kinds of combinations of sounds in advanced tuberculosis.

Respiratory Rhythm.—The normal relationship of inspiration to expiration is disturbed in such a manner that as infiltration increases expiration gradually lengthens until it finally becomes equal to or longer than inspiration. The change in rhythm is usually accounted for in tuberculosis by the consolidation causing the bronchi to be more rigid and the tissues less elastic, as a result of which the air is forced out of the lungs more slowly and the sound emitted transmitted more readily. So it can be understood that all degrees of prolonged expiration exist in tissues the character of which varies from slight infiltration to marked fibrosis or cavities and dilated bronchi surrounded by dense fibrous tissue. I do not doubt that the atrophy of the soft tissues external to the pleura also has its influence by removing a compressing influence on the thorax, thus prolonging the expiratory phase. When compensatory emphysema occurs, prolonged expiration is favored by the lessened elasticity of the pulmonary tissue failing to expel the air; but the quality of the note differs wholly because it lacks the conditions for increased transmission. While the expiratory note of consolidation and scar tissue is more intense and higher in pitch than normal, that of compensatory emphysema is lower pitched though it may be louder than normal.

Quality of Note.—The quality of the respiratory note also varies greatly. As the pathological changes in the lung extend, the soft breezy normal inspiration gives way to a rough, harsh, or combined rough and harsh, inspiration with pitch higher than normal. The same is true of expiration. Expiration, as a rule, is equal to or of higher pitch than inspiration when consolidation is present; and particularly in the presence of dense fibrous tissue. When excavation has occurred, however, there is a tendency for the pitch of the expiratory note to be lower than that of inspiration, unless the cavity is small and surrounded by large amounts of dense fibrous tissue. This is an important sign of cavity.

The roughness which so often characterizes the respiratory note in advanced tuberculosis probably has many factors which enter into its composition. It has been suggested that roughness in early tuberculosis may be caused by an unequal entrance of air into the tissues. The condition which would produce roughness because of such unequal entrance, is found to an exaggerated degree in advanced tuberculosis, because of the varied pathological processes which are present. In advanced tuberculosis there is often found coexisting in the same lung, or even the same lobe, varying degrees of infiltration accompanied by varying degrees of exudation, areas of necrosis, excavation and fibrosis.

The effect of the muscles in producing the roughened character of the note must also be considered. The peculiar rough, rumbling respiratory note over compensatory emphysema undoubtedly has a large muscular element in its production. Thickenings of the pleura which are stretched during respiration also add a roughened character.

Changes in the respiratory note in advanced tuberculosis consist in replacing the normal vesicular murmur by one which, at first, is somewhat bronchial in character, but which, later, after pathological changes become sufficiently extensive, becomes entirely so. This, in turn may become amphoric when excavation occurs.

Râles.—Râles or adventitious sounds accompany the respiratory murmur in nearly all cases of widespread pulmonary tuberculosis. These râles vary in nature as they differ in the manner of their production. Râles are divided into moist and dry. Some writers claim that all intrapulmonary râles originate with moisture. At times stretching of scar tissue, pleural adhesions and muscles will produce sounds that cannot be differentiated from the intrapulmonary sounds. The methods of differentiating intrapulmonary and extrapulmonary râles is uncertain.

Râles are affected by the depth of respiration, cough, position of the patient, whether the patient has coughed and expectorated immediately prior to examination or not, the time of the day, fever, conditions of weather, and many other factors, as well as the pathological process which produces them.

By making frequent examinations of a tuberculous lung over a prolonged period of time it will be noted that the râles are characterized by constancy. When râles appear in an area they go through certain evolutionary changes but do not leave the part wholly until healing or wholesale destruction with cavity formation has taken place and not always then. They might be temporarily put in abeyance by a pleural exudate or pneumothorax, but they do not disappear until the cause disappears. Many râles remain permanently, even after an apparent healing has occurred.

Râles, to a certain extent, indicate the nature of the pathological process underlying. Probably, if we were more expert in our interpretation, they would be even more accurate. The first râles which make their appearance are those which accompany infiltration, the so-called dry crackle or *crepitant râles.* They are found in the areas which have been recently affected. At this time the process is accompanied by very little exudation into the tissues surrounding the tubercles. The râles are confined to the finer air passages, largely to the air cells. The result is that the râles are fine and dry in character and are elicited best by increased inspiratory effort or by this preceded by a cough.

In auscultating over early active infiltrations, these râles are often heard in small showers after coughing. In more advanced lesions in which the bacilli are multiplying, tubercles are softening and toxins are diffusing into adjacent tissues, a greater degree of inflammation exists and this is accompanied by increased exudation. This process is accompanied by increased moisture in the air passages which finds its way into the bronchi of different sizes and in this way produces moist râles, both medium and large in character.

In localized areas of necrosis with cavity formation medium or large râles may persist for a long time. The location of conglomerate caseating tubercle is often marked by the persistence of medium and large râles heard over a given area for a considerable time. If extensive excavation takes place the râles may disappear wholly, or decrease in number coincident with the sloughing of the tissue. Where one has been examining patients repeatedly, this cavity formation may be suspected by the absence of medium and large râles which have previously existed for a long time. On the other hand, there are times when, coincident with such pathological changes, the râles become coarser and bubbling or gurgling in character, owing to the air forcing its way through the mucus or pus which is present in the cavity.

When healing is taking place, the multiplication of bacilli in the tubercles is lessening, and the amount of toxins diffusing into the adjacent tissue is decreasing, consequently the exudative inflammation which accompanies active inflammation decreases and the amount of moisture which shows as moist râles, lessens and the tissues appear to the examiner to be of a dryer character.

This statement must not be considered as conflicting with the fact, which is often evident in the fibro-ulcerative type of tuberculosis, that the amount of sputum does not necessarily lessen in the proportion to the lessening of râles. The explanation is that the sputum is coming from ulcerative surfaces within cavities instead of from the bronchial mucous membrane.

Not only do we have the three types of distinctly moist râles just described, but there are squeaks, wheezes, and rhonchi of great variety. Often a persistent squeak at some point in the pulmonary tissue indicates that softening and ulceration is taking place. Wheezes and rhonchi are at times present in such numbers that the picture resembles asthma.

Adventitious Sounds Resembling Intrapulmonary Râles.—The examiner is often much confused in knowing how to interpret the adventitious sounds which are heard upon auscultation. I have just described many different types of râles that have their origin within the lung. These râles would seem to be more or less distinctive of certain conditions which produce them. There are, however, many sounds heard on auscultation, which cannot be readily differentiated from the intrapulmonary râles just mentioned. Pleural crepitations are, at times, very difficult to differentiate from intrapulmonary râles. The muscles also, now and then, give us sounds which cause difficulty in diagnosis. Sometimes these are fine crepitations, sometimes they simulate medium, and, at other times even coarse râles; or, they may even take the form of creaks and squeaks. We find many of these near the apex where the pleura becomes thickened, and the adjacent musculature and tissue also partake in the inflammatory process, as has been described by Coplin (see Volume II, Chapter XXV). These râles may be found in any part of the lung where a thickened pleura and chronically inflamed muscles exist.

Swallowing will often produce sounds which simulate râles, when one is listening over the apices. After the patient has been observed for a long time, the true nature of these râles can usually be made out by the fact of their persistence and the knowledge of the changes which have occurred in the underlying pulmonary tissue.

Where widespread pleural adhesions are present we may often infer that the râles are not intrapulmonary by the atrophied overlying soft tissues and by the fact that the amount of moisture which would be represented by such widely distributed pulmonary râles, is not in keeping with the amount of sputum present.

Extensive Infiltration in One Lung.—In discussing the more common conditions which are met in the lung in advanced pulmonary tuberculosis I shall first consider extensive infiltration.

Inspection.—What can be determined by inspection will depend, as previously mentioned, on whether the infiltration in the lung is recent or of long-standing, and upon whether it is a new infiltration in tissue which has not previously been infected, or a renewed activity in an old lesion.

No matter whether the infiltration is a new or old one, if it is active, it causes a limitation of the respiratory movement of the chest wall. All active inflammations of the pulmonary parenchyma cause reflex motor disturbance in the muscles of respiration which results in limited motion. The diaphragm, being the chief muscle of respiration, and being affected by this reflex, acts in such a manner as to greatly alter the respiratory movements. Interference with the elasticity of the pulmonary tissue, likewise causes diminished motion. Sometimes this limited motion is most pronounced at the apex. The apex is the usual site of the first acutely active tuberculous process in the lung, and when the disease is extensive, it is nearly always accompanied by pleural adhesions which also limit the respiratory motion. At other times, limited motion is most marked at the base; or, again, it may be equally distributed over the entire side.

Limited motion is best detected, as a rule, during normal easy respiration. At times, however, it is best elicited on deep inspiration. Examination should be made during both shallow and forced respiration.

The changes in the soft parts are often plainly visible, as described in discussing early tuberculosis in Chapter XV. In early tuberculosis, increased tone (spasm) of the muscles is of the greatest diagnostic significance. In advanced tuberculosis, as a rule, degeneration is most evident and of greater diagnostic importance than increased tone. This will vary, however, according to the activity of the pathological process; whether it involves tissues which have not been previously infected; or is an increased activity in an old focus. This latter is the condition usually met in advanced tuberculosis.

When a disease has existed in the lung for a prolonged time and become chronic in character, the soft tissues of the neck and chest above the second rib anteriorly and the spine of the scapula posteriorly, show degeneration. If in the pleura the degeneration is over the ribs. Consequently, degeneration of the soft tissues covering the chest, when regional in character, becomes a very important sign of chronic inflammation in the underlying pulmonary or pleural tissue.

If the inflammation is recent, aside from the degeneration, the muscles will show increased tone. This, at times can readily be detected on inspection, particularly in the sternocleidomastoideus, trapezius and levator anguli scapulæ.

If the process has been chronic and the muscles have accordingly degenerated, then it may be difficult or impossible to determine the increased tone on inspection; in fact, such an increased tone may be, now and then, especially in individuals with weak musculature, difficult to determine with certainty on palpation.

Palpation.—Palpation shows increased tone of the muscles covering the apex and upper portion of the lung if the disease is active and of recent extension. Degeneration of these same muscles, skin and subcutaneous tissue takes place when the disease is of long duration. This is shown by the tissues presenting a soft, doughy, inelastic sensation to the palpating fingers, and by the fact that the cushion of subcutaneous tissue, likewise the mass of muscles is smaller in amount than normal. Increased tone may be noted in the degenerated muscles if the disease is chronic, but still active. At times, however, the degeneration will be so marked that the increased tone may not be detected. Even those with most practice may have difficulty in being sure of the increased tone under these circumstances. I would call attention to the fact, however, that the sternocleidomastoideus, trapezius, and levator anguli scapulæ should be examined most carefully under such conditions, for they will usually reveal the true condition of the muscles in general.

Palpation corroborates the lessened excursion of the side involved. As a rule, this important sign can be determined better by palpation than by inspection. The hands should be laid on lightly, however, for even slight pressure will restrict the movements.

Through palpation we also determine the relative density of the lung tissue. If one is endeavoring to determine the areas of infiltration, it is better to palpate from areas of lesser density to those of greater density; consequently, in palpating lungs it is preferable to begin at the base and go upward, unless thickened pleura interferes. When the borders of the areas of infiltration are reached, an increased sense of resistance will be noted by the palpating finger. One must be on his guard in palpating the total resistance of the lung because there are other factors than the thickened pulmonary tissue which enter in and have a tendency to vitiate the findings, the same as they do in percussion. For example, in palpating in the axillary line, when one comes to the folds of the pectoral muscles an increased resistance is noted. This must not be considered as being necessarily due to the pathological changes in the lung. It must be considered in connection with the fact that the more tissue through which one palpates, the greater the resistance to the fingers. Another area which offers difficulties is the interscapular region. An increased feeling of resistance is recognized by the palpating fingers when the lower border of the rhomboidei muscles is reached. This may be mistaken for infiltration. These same muscles give difficulty, and often mislead, in percussion the same as in palpation. Infiltrations in the lung are detected as readily by palpation as by percussion.

If the existing infiltration is sufficiently extensive to call for compensatory changes in other portions of the lung, or in the opposite lung, a lessened total density may be noted over the emphysematous portions. This lessened total density is readily determined by comparing it with the increased deep resistance over the area of infiltration. Palpating over marked emphysema, however, often gives an increased resistance to the palpating finger, which may be mistaken for increased density in the underlying pulmonary tissue. This is due to the fact that marked emphysema causes an increased tension of the intercostal muscles which shows as increased resistance to the palpating finger. If, under such circumstances, however, one will palpate more deeply he will readily see that he is palpating over increased tension, but not over increased density.

Percussion.—Percussion will give results according to the state of the tissues percussed. The examiner must bear in mind, as mentioned in connection with percussion in early tuberculosis (page 420), that the percussion note and the degree of resistance conveyed to the finger are influenced not only by the state of the underlying pulmonary tissue, but by the pleura, bony thorax, subcutaneous tissue and muscles. If the muscles are hypertrophied from use or increased in tone (spasm) because of the reflex stimulation from the pulmonary inflammation, the percussion note will be higher and the resistance greater than normal. If they and the subcutaneous tissue are atrophied from reflex trophic changes or from disuse following hypertrophy, then the note will be more resonant and the resistance less than normal. Thickened pleura will also cause higher pitch of the note and greater resistance to the finger.

An infiltrated lung may show varying degrees of infiltration, each of which will give a different character to the percussion note, even though the coverings of the lung are always the same.

These facts show that there can be no definite note or degree of resistance which will always mean a given degree of infiltration in a lung. They show too that the percussion cannot be accurately interpreted without estimating the effect of the changes in the coverings of the lung, particularly the amount and the degree of increased tone or wasting of the muscles; the amount and the degree of wasting of the subcutaneous tissue, and the amount of thickening of the pleura, when present. Failure to appreciate this is the cause of much error.

Oftentimes an infiltration of greater density than really exists will be indicated by the higher pitched note or greater resistance to the finger on percussion which results from the reflex increased tone (spasm) of the muscles overlying the lung. While this condition is most often met in early tuberculosis, yet it is not infrequent in advanced cases when the disease has spread quickly. This is particularly apt to occur over the trapezius, levator anguli scapulæ, and rhomboidei; and, at times, also over a well-developed pectoralis. On the other hand, an infiltration of lesser degree than actually exists may be diagnosed when these same muscles and the subcutaneous tissue overlying them have wasted. This is most pronounced when the wasting is a reflex trophic change. At times, from reflex trophic changes, the soft parts on one side of the chest will be reduced fully one-third in mass as compared with the same muscles and subcutaneous tissue on the other side. The effect of this upon the data derived by percussion is considerable.

The first extensive infiltration is very often in the lung opposite to the one which was the seat of the original focus; consequently the condition of the soft tissues is extremely important. Over the lung with the old lesion, there is wasting of varying extent and varying degrees, while over the area of the newer infiltration, unless it has existed for many months, there is increased tone (spasm) of the muscles.

Bearing all these alterations in mind, and they are extremely important in advanced tuberculosis, we find the following conditions on percussion in extensive infiltration.

If the tubercles are scattered over a considerable lung area, but not thickly set, they do not cause the amount of thickening of the tissue necessary to produce marked dullness; but interfere with the normal elasticity, cause more or less relaxation of the pulmonary parenchyma and produce a note tympanitic in character.

If the infiltration is more dense and particularly if the infection is of such virulence as to produce much exudation in the tissues, then we have varying degrees of dullness according to the mass and virulence of the infiltration and the character of the exudation, leading up to the flatness of caseous pneumonia; and varying degrees of resistance to the finger reaching its maximum also in those cases which show the thickest studding of tubercles and the greatest amount of exudation.

One condition which is extremely confusing is an infiltration which takes place in the second lung when it has been the seat of a high degree of compensatory emphysema because of a widespread destructive process in the other lung. Here the fundamental findings on percussion are those of emphysema,—increased resonance and decreased resistance to the finger as compared with the normal. If the tension is extreme, however, and the chest bulging and the intercostal muscles stretched, the note may be somewhat tympanitic or dull and there may be a sense of increased resistance to the finger. This condition, while readily detected by the expert examiner, at times proves confusing to those of less experience. It can readily be seen that infiltrations of slight or even moderate degree, when existing in such an emphysematous lung, might be overlooked on percussion, being overshadowed by the findings due to the underlying emphysema.

Infiltrations underlying thickened pleura are extremely difficult to make out on percussion because of the changes incident to the altered pleura.

Auscultation.—Auscultation over infiltrated lung tissue shows a variety of sounds. Râles may or may not be present. Extensive infiltrations may exist without râles. This fact is not sufficiently appreciated. Too much dependence in diagnosis is placed upon the absence or presence of râles, for it is a well established fact that there is a form of tuberculosis of low virulence which extends over wide areas, irritating the cells and causing new tissue formation, the entire process taking place without any marked exudation accompanying it, or without ulceration and loss of tissue. Auscultation over such areas shows a respiratory note often somewhat weakened; harsher than normal, and usually accompanied by a prolongation of the expiratory note. Roughness is also a frequent characteristic if activity is present.

If the process is more virulent, other pathological changes occur. The bacilli multiply, necrosis occurs, toxins diffuse into adjacent structures, and exudation takes place. Such infiltrated areas show, on auscultation, aside from the characteristic harshness, roughness, and prolongation of expiration, a variety of accompanying râles. These may be fine, medium or coarse; dry or moist, according to the conditions surrounding their production.

Fibrosis.—Fibrosis occurs in tuberculosis when the infecting bacilli are of a low grade of virulence. It is a result of irritation whereby the tissues are stimulated to new growth. The cells multiply and the tissues thicken, increasing their density. Healing in tuberculosis results in the formation of fibrous tissue. When ulceration has occurred, however, other conditions will also be present. The formation of fibrous tissue is, as a rule, a slow process, and the changes found on inspection, palpation and percussion are much the same as those of long-standing infiltration. Consequently *inspection* and *palpation* show the degenerative changes in the muscles and subcutaneous tissue, often with diminished motion of the side. Increased density is also noted by palpating over the fibroid areas; and, if the process is extensive, a compensatory emphysema is usually present in other portions of the lungs not involved in the fibrosis which affords a decreased resistance to the palpating fingers.

Percussion shows a higher pitched note with a degree of dullness and increased resistance to the finger which are commensurate with the amount of fibrosis present; and a note more resonant than normal with lessened resistance to the finger over the portions of lung tissue which are the seat of compensatory emphysema when this is present.

Auscultation reveals varying degrees of harshness, often high pitched in character, with prolonged and often high pitched expiratory note. The latter is often of a blowing character and may simulate cavity. A point of differential value is that the blowing expiration in the presence of cavity is usually lower in pitch than the inspiratory note while that which accompanies fibrosis is higher. The note over fibrosis may also be accompanied by adventitious sounds. Sometimes these arise in the pleura, sometimes in the fibrous tissue itself and at times in the muscles.

Cavity.—Cavity results from caseation and rupture of tubercles. Cavities may be as small as pinheads or so large that they occupy an entire lobe or entire lung. At times no single large cavity but many small ones are present. Often several large communicating cavities exist. Cavities may have smooth dry walls or they may be ragged and secreting. The more chronic cavities, as a rule, have the smoothest walls. Cavities may be surrounded by scar tissue, or infiltrated tissue in any stage of pathological change to which tubercles are subject. The same cavity may at one time contain secretion, at another be empty. It can readily be seen that these various conditions cannot produce the same physical signs. Often one sign will be present on one examination and be absent at another. This must be understood in order to avoid confusion. The history is of great value in determining whether or not cavities are to be suspected. From history alone one can usually determine whether loss of tissue is to be suspected. Fever, accompanied by cough and free expectoration, has nearly always been present at some previous time, even if not at the time of examination, if cavity is to be suspected. If the patient is expectorating more than 20 to 30 c.c. of sputum, unless an acute or chronic simple bronchitis is present, cavity is to be suspected. In chronic cases where healing is occurring cavities may be found when the amount of sputum is decidedly small.

Inspection.—There are times when the musculature and subcutaneous tissue on the front of the chest have wasted to a marked degree and superficial cavities may be suspected by the sinking in of the soft parts during respiration. This, however, is not sufficiently reliable for diagnosis.

Palpation.—Loss of tissue can often be readily detected on light touch palpation. In palpating over a tuberculous lung, infiltrated areas will be recognized by a degree of resistance to the finger, which is greater than normal. If a cavity, or several cavities are present which are alone or together sufficiently large to represent a considerable loss of lung tissue, they are detected by an area of decreased resistance noted by the palpating finger in the midst of the increased resistance which belongs to the surrounding infiltration. I have learned to rely on this sign—*a resistance less than normal noted on palpation in an area surrounded by resistance greater than normal*—as being the most constant evidence of loss of pulmonary tissue. This sign is present whether there is one large, or many small, or several communicating cavities. It is also present at all times unless the cavity be filled with secretion.

Percussion.—Percussion over cavities shows several changes from the note elicited over surrounding tissues. It may be impaired, more resonant, tympanitic, or the pitch may be lower, or it may show the "cracked pot" or Wintrich phenomena. The character of the note depends much on the force of the percussion blow. There is no particular percussion sound or phenomenon that is found with regularity over cavities. Increased resonance results from the loss of tissue and can usually be elicited, if the stroke is not too forceful and the cavity is sufficiently large. The note, however, is modified greatly by surrounding tissue, so the sound which particularly belongs to the cavity can best be elicited by a light stroke.

To my mind the most characteristic note found over areas of excavation which are surrounded by infiltration, is a "dull tympany" elicited on a stroke of moderate force.

"Cracked pot" may be heard over superficial cavities with smooth walls when freely communicating with a bronchus. Most definite "cracked pot" sounds may be heard at one examination and be absent at another a few hours later, because of a closure of the cavity opening. Wintrich pointed out that the tympany over cavity assumes a higher pitch when the mouth is open. An absence of these signs must not be taken as having any bearing on the presence of cavity. Resonance over cavity is sometimes relatively greater because of marked wasting of soft tissues (muscles and subcutaneous tissues) which would otherwise add a dull character to the note.

The lack of resistance to the finger is of greater importance than the character of the percussion note. The increased resistance noted over the infiltrated area will be absent when excavation has occurred, as mentioned under palpation. This condition in the midst of dullness can mean but one thing—a lack of tissue underlying, and while this might be due to a dilated bronchus or a circumscribed pneumothorax as a rule it means cavity.

Auscultation.—Auscultation over cavities may reveal undoubted proof of their existence or questionable or negative information. Where the examiner has been listening at frequent intervals over a considerable period of time over areas the seat of widespread infiltration and caseation, he will now and then find that the râles have suddenly disappeared and the sounds have become clearer. This does not mean that the tubercles have become less active and that collateral inflammation has disappeared and that cicatrization is progressing; but it means that the tissue has sloughed out leaving a cavity behind. An acute cavity may be suspected at times on this finding alone.

Chronic cavities may also be suspected by an absence of râles in the presence of large quantities of sputum. If the sputum comes from disseminated areas of infiltration and necrosis in the pulmonary tissue, medium and large-sized râles will be heard in abundance over the entire area affected; but, if the tissues are free from râles, this shows that the sputum must come from elsewhere, a cavity or cavities, empyema with bronchial fistula being the only alternative. Not infrequently do we find patients suffering from chronic inactive tuberculosis who expectorate 50 to 100 c.c. of sputum daily, but who show few râles on examination. On the contrary coarse bubbling râles, localized and persistent, often suggest cavity. These may be metallic in character, particularly if the cavity is large and possessed of smooth walls.

If cavities are superficial and covered by thickened pleura, or if they are surrounded by scar tissue, aside from the râles which originate within the cavity, many adventitious sounds are at times heard. These may simulate medium râles, but, as a rule, are dry in character and heard both on inspiration and expiration.

The breath sounds heard over cavity may be changed very little, if any, from those over the surrounding tissue. Sometimes the sounds are diminished, this particularly when some interference with the ingress of air exists.

Blowing expiration is frequently found over cavities, but is not pathognomonic. This character of breathing is also found at times over scar tissue, particularly near the apex. I am influenced very much in my decision as to whether the blowing is due to cavity or scar tissue by the relative pitch of the inspiratory and expiratory sounds. If expiration is higher pitched than inspiration the blowing is most apt to be due to fibrosis; if it be lower pitched it suggests cavity.

Amphoric breathing is occasionally found, but only in a small percentage of cases; and only at times in these. A peculiar distinct echo on coughing is sometimes transmitted to the stethoscope. While this is not regularly heard, it is characteristic of cavity.

A study of whispered voice transmission helps to detect cavity. Such syllables as "one," "two," "three," "ha," "ha," or "whisper-r-r-r" are more distinctly transmitted as syllables over cavity than over infiltration or fibrosis, and when considered in conjunction with other signs are of value in diagnosis. A cavity large enough to give signs must be about as large as a walnut. Several smaller ones in groups, however, may be detected. As a result of the compensatory changes within the thorax small cavities may be compressed and disappear; and large ones may be so compressed as to completely alter the signs and symptoms caused by them. An alteration or disappearance of signs after once being found must not be taken as indicating that the previous findings were incorrect.

Compensatory Emphysema.—Compensatory emphysema is a part of practically every advanced case of pulmonary tuberculosis that reaches the stage of loss of tissue, whether through new tissue formation and contraction, or through destruction and cavity formation. I look upon it as being an expression of nature's attempt to equalize the atmospheric pressure within the lung and that upon the surface of the chest as described more fully on page 282.

Compensatory emphysema is a dilatation of the air cells. In tuberculosis it affects the tissues which are most free from infection, those which are most useful to the patient. Where one lobe is the seat of cavity or marked fibrosis, a marked degree of compensatory emphysema will usually be distributed over a considerable portion of the remaining pulmonary tissue. If both upper lobes are the seat of destructive change, the bases are emphysematous; and if one entire lung is involved in the destructive process the other lung becomes emphysematous. Emphysema distorts the lobes and displaces their boundaries, as illustrated in Fig. 40, page 286.

This condition is sometimes spoken of as an hypertrophy of the lung, rather than an emphysema. It is not primarily an emphysema in the sense that it is a degeneration of the alveolar walls, neither is it an hypertrophy in the sense of it being an increase in tissue, such as we see in the heart muscle for instance. I very much doubt if it is wholly what we have taught—an enlargement of that portion of the lung which is still able to functionate as a result of nature's effort to compensate for the parts destroyed—but look upon it as a compensatory enlargement of pulmonary tissue in order to fill the thoracic cavity. It occurs in consequence of an attempt to preserve an equilibrium between the atmospheric air as it presses upon the surface of the chest and as it presses on the air cells from within through the trachea and bronchi. The result is a dilatation which to all appearances is an emphysema. The extra work thrown upon it is due to the loss of tissue, but it is not a cause of the dilatation of the air cells. If the compensatory change is to be looked upon as an attempt on the part of the lung to better carry out its function, the emphysema is a mistake because the dilated air cells are not functionally as capable as the normal cells.

Inspection.—It can readily be understood that compensatory emphysema, with its distended air cells and its pressure on surrounding structures, will, if well marked, cause alterations in motion and bulging of the thoracic walls from which its presence may be suspected. If of slight degree no change may be noted on inspection.

By carefully inspecting a chest which is the seat of marked compensatory emphysema one can often form a fairly accurate impression of the pathological changes within the lungs. In generalized emphysema in lungs which are not the seat of destructive processes, the anteroposterior diameter of the chest increases, the lower ribs become elevated, the costal angle widens, the diaphragm becomes displaced downward, and the motion of the thoracic walls is decreased. Compensatory emphysema on the other hand is confined to limited portions of pulmonary tissue, because there is a decrease in the total pulmonary area. The air cells which are not destroyed are involved in the emphysema. They are dilated, and yet, in spite of this, they are compelled to aërate the blood. Consequently, all inspiratory effort is directed toward the emphysematous areas as being the parts which are not destroyed; consequently these portions of the chest often take upon themselves an increased movement. Localized areas of bulging with increased motion are very suggestive of compensatory emphysema. When one lung is the seat of marked destruction and the other is markedly emphysematous, the auxiliary muscles of respiration are called upon for extra work and so stand out plainly on inspection. This is particularly true of the sternocleidomastoideus and scaleni. Under such circumstances, too, we sometimes see the supraclavicular fossa swell up on forcible cough. One must not mistake the increased tone in the neck muscles in the presence of emphysema for reflex muscle spasm.

Palpation.—Palpation over areas of compensatory emphysema shows tense intercostals, increased tension of that portion of the lung affected, and usually increased movement. The total density, as determined by the resistance to the palpating finger, is less than normal, where the pleura is normal and less than would be expected where covered by thickened pleura, or where previously or secondarily the seat of infiltration. Palpation shows the borders of the emphysematous lung to be pushed outward. Many times the diaphragm and mediastinum are pushed far from their normal position, as described on pages 285 and 305.

Percussion.—Percussion shows hyperresonance, and where intrapulmonary tension is high, tympany; although these sounds are greatly modified by other conditions present, such as thickened pleura and infiltrations. Lessened resistance is also present, less than that over normal lung when neither infiltration nor thickened pleura are present.

Auscultation.—Emphysema shows many changes in the respiratory sounds. Sometimes the note is weak; but this is more apt to be the case in generalized emphysema than in the compensatory form. When the note is weak in compensatory emphysema it is usually because of thickened pleura, for the note is usually exaggerated because of the extra work thrown upon the part.

The characteristics of the respiratory sound in compensatory emphysema are a roughened inspiratory note which often partakes of or gives way to a harshness as the inspiratory effort becomes more intense and the note becomes more exaggerated; and a prolongation of the expiratory note due to the slowness with which the emphysematous lung completes the expiratory phase. The prolonged nature of the expiratory note is often overlooked by not listening intently through the entire expiratory phase of respiration.

It has often seemed to me like the contraction of the tense thoracic muscles has much to do with the character of the inspiratory note in this affection. In this connection Kingston

Fowler[2] states, in discussing emphysema in general: "In place of this normal vesicular murmur audible on inspiration, the continuous low pitched rumbling sound produced by the contraction of the muscles is very distinct."

Dry Pleurisy.—Dry pleurisy is a frequent accompaniment of advanced tuberculosis. It comes at times as a localized process accompanying an area of caseation near the pleural surface; again, it manifests itself as an acute inflammation more or less widespread involving the subpleural tissues or pleural surfaces. Either process may result in adhesions.

Inspection.—A marked limitation of movement on the side of the inflammation is usually evident to the eye, particularly if the process is near the bases.

Palpation usually detects an increased tone (spasm) of the intercostal muscles which often feel decidedly boardy. It may also detect a pleural rub. It likewise confirms the limited motion noted on inspection.

Percussion is practically valueless.

Auscultation will reveal moist râles over the pulmonary focus when the pleurisy is due to active caseation of a subpleural pulmonary focus. It will also detect a pleural rub or roughness when the surfaces of the pleura are inflamed. A grating sound may be heard at times for some time after the acute symptoms disappear. Pain is usually a symptom and of itself is often sufficient for diagnosis.

Pleural Effusion.—Pleural effusion now and then complicates advanced tuberculosis. This complication is found comparatively infrequently in patients who are cared for properly. It seems that there is something in the general tonic treatment especially when fortified by tuberculin that makes effusions rare. The symptoms and physical signs vary with the size of the effusion.

Inspection.—Effusions when large may be suspected or even diagnosed by the bulging ribs and interspaces on inspection. Dyspnea may be present. The bulging of the ribs, limited motion, and displacement of the heart to the opposite side in the absence of symptoms pointing to pneumothorax, suggest effusion.

Palpation.—Palpation shows increased resistance over the fluid due partly to the increased tension of the intercostals and partly to the increased density caused by the fluid. The outlines of the fluid can be detected by light touch palpation as readily as by percussion. Voice transmission as determined by palpation and auscultation is diminished. Palpation also confirms the limitation of motion on the side.

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[2] Allbutt Rolleston: System of Medicine, vol. v, 1909, Macmillan Company, Ltd., New York.

Percussion.—Percussion may show any condition from a slightly impaired note to a very dull one. It also reveals increased resistance to the finger. The data obtained, however, requires confirmation and must be considered in conjunction with that obtained by other methods of examination. Groco's sign, which is a triangular area of dullness at the base near the vertebral column on the side opposite the effusion, usually may be found. It is most pronounced and easiest of detection when the effusion is large.

Auscultation.—The data obtained on auscultation over the effusion may be nothing more than a weakened respiratory note; but this is of value in conjunction with other data. There is apt to be an exaggerated note over the pulmonary tissue above if the effusion is large.

Thickened Pleura.—The pleura may show any degree of thickening from one that is barely recognizable to one of several centimeters. This is an extremely common condition in pulmonary tuberculosis. It is rarely that we find apical pleuræ free; for, as the disease affects the surface of the lung the irritation extends to the pleural surfaces and adhesions and thickenings result. Adhesions are favored at the apex by the limited motion of the part. Likewise they are opposed at the bases by the extensive movement. When, through disease in the underlying lung or of the pleura itself, the respiratory movements are greatly limited, adhesions are favored. After adhesions have once taken place increased thickening is probably favored by the irritation resulting from the movements of the thoracic wall and their pull against the adherent pleuræ.

Inspection.—Inspection usually shows a limited motion which is most marked when the thickening extends to the base; but this differs in no way recognizable on inspection from limited motion from other causes, except, at times, the side seems to move as a unit. The wasting of the soft tissues over areas of thickened pleura of long duration is often plain to the eye.

Palpation.—Palpation gives the most reliable information that we have in determining thickened pleura. It not only confirms the lack of motion but it reveals an increased density of the palpated tissues varying with the degree of thickening, and shows by the peculiar characteristics of the soft tissues (skin, subcutaneous tissue, and muscles) overlying the pleura that there is a pathological condition underlying. Degenerative changes take place in the overlying muscles as described by Coplin[3] which give very valuable diagnostic data on palpation. (See Volume II, Chapter XXV.) The soft tissues overlying the thickened pleura lose their elasticity and feel lifeless and doughy. The tissues feel to the touch somewhat like those affected by a very slight edema. Later the subcutaneous tissue and intercostals degenerate and at times contraction takes place, drawing the intercostal spaces nearer together. Bönniger[4] has described a unilateral lymph-stasis as affecting the skin, subcutaneous tissue, and muscles over the diseased pleura. He states that the tissues appear to be increased in volume and firmer than normal. He mistakenly offered this as an explanation for the spasm of the muscles which occurs in the presence of active disease in the pleura and lung. In this he undoubtedly recognized the doughy, inelastic, lifeless condition which I have described.

Percussion.—Percussion alone gives nothing distinctive of thickened pleura. It reveals by a higher pitched note, a slightly decreased resonance and an increased resistance to the finger that there is an increased density of the tissues underlying. Which tissues are responsible for the changes can only be determined by considering the data, together with that obtained by other methods of examination.

Auscultation.—Auscultation over thickened pleura will show varying degrees of diminished breathing; but since this complication is usually associated with infiltration of the underlying pulmonary tissue, and since this of itself often causes diminished breathing, a weakened respiratory note must not necessarily be considered as due to thickened pleura. Other signs such as those determined on palpation should be looked for. Sometimes when the pleura is thickened there are many adventitious sounds present which accompany both phases of the respiratory act. They may sound like medium râles, but, for the most part, are dry in character. They are apt to start with inspiration and continue until the end of expiration; and they give the impression of being near the stethoscope. Where these originate is not definitely settled but we are safe in saying that they originate either in the muscles, subcutaneous, or pleural tissues.

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[3] Changes in the Intercostal Muscles and the Diaphragm in Infective Processes Involving the Lungs and Pleura, American Journal of Medical Sciences, May, 1904.

[4] Bönniger: Über halbseitige Lymphstauung bei Erkrankungen des Lunge bzw. Pleura, Berliner klinische Wochenschrift, Nr. 25, June 20, 1910.

Mediastinal Thickening.—The mediastinum is a region which has been far too much neglected in clinical medicine. Prior to the use of the x-ray it was considered almost impossible to diagnosticate pathological conditions in this part of the thoracic cavity, except they were of the most evident type. But, after obtaining a more definite idea of the processes which affect the mediastinum most commonly, we are now, clinically, either with or without the aid of the x-ray, able to examine this field with a fair degree of satisfaction.

The mediastinum is a fruitful field for study in tuberculosis and particularly in advanced tuberculosis. It at times shows fibrous thickening which is important in the causation of symptoms even when the disease of the pulmonary tissue has not been suspected. Enlargement of glands, which often belongs to the stage prior to the pulmonary involvement persists and is evident on careful examination in from 20 to 25 per cent of cases. Adami and MacRea[5] say that one-fourth of all healed cases of pulmonary tuberculosis show enlarged bronchial glands. That type of tuberculosis which starts at the hilus and extends out into the pulmonary tissue really belongs primarily to the mediastinum. Tumor masses of non-tuberculous nature, either solid or aneurism, at times complicate tuberculosis and must be looked for. Aside from these thickenings, the shifting of the mediastinum is extremely important and deserves careful study. (For a more complete description of this see page 285.

Fibrous thickening of mediastinal tissue, particularly with marked tracheal shifting, as it occurs at times in advanced pulmonary tuberculosis, in my experience, is accountable for several direct symptoms, differing according to the nature of the case.

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[5] Transactions of the Sixth International Congress for Tuberculosis, 1908.

In a few cases hoarseness is a marked symptom; depending on the extent to which the laryngeal nerves are irritated. Cough, coming on in asthmatic paroxysms, is not uncommon. It may have a brassy tinkle. Bronchitis is often a common accompaniment and is usually of a stubborn nature.

Inspection.—As a rule inspection shows nothing definite as to thickening which occurs within the mediastinum. Chronic inflammation shows by atrophy of the interscapular soft tissues. Occasionally, tumors, if large, may be suspected, by bulging of the thoracic wall.

Palpation.—Palpation and percussion are our most valuable measures in diagnosis. By palpating over the sternum and vertebral column one will learn the usual degree of resistance which marks the normal mediastinum; and will be able to detect any increased resistance due to pathological changes.

New tissues, when present in the mediastinum, may belong to the tissues normally there or to pathological processes extending out from the pulmonary parenchyma; and the findings will vary accordingly. Anteriorly, the feelings of increased resistance will be under the sternum in the former case, while they will push out beyond its borders in the latter. It is well after palpating the sternum, to commence out in the interspaces and gradually approach the sternum, then repeat the same over the ribs. Any resistance should be noted. On numerous occasions I have been able to suspect pathological deposits of fibrous tissue adjacent to the hilus which were confirmed by the x-ray, simply by noting the departure from the normal in the resistance to the palpating finger. Posteriorly, by straddling the spinous process with the first and second finger, as shown in Fig. 16, page 116, and, beginning above, palpating downward, one will learn to detect abnormally increased resistance, if present. Usually, if due to peribronchial and peritracheal glands, it will be detected at the level of the third dorsal vertebræ. This increased resistance as a rule continues into the heart resistance, which normally begins in the average adult with the sixth dorsal. The resistance is nearly always greater on the right than on the left side of the spine because of the localization of the trachea on that side. It must not be forgotten, however, that a certain amount of shifting of the mediastinum is common in advanced tuberculosis and that it affects the position of the glands.

Percussion.—Percussion also shows the increased resistance to the finger though probably it is not as sensitive as palpation. Frederick T. Roberts[6] recognizes the value of resistance in percussion as follows: "It is highly important in relation to mediastinal tumors to bear in mind when carrying out the method of percussion, not only the sounds which are elicited, but also the sensations which are felt by the fingers during the act. Not uncommonly the latter afford most valuable information." Personally, while practicing both, I deem the resistance of greater value. Percussion over mediastinal thickenings will usually show an impaired note. It may be of any degree of dullness or even flat, according to the amount and nature of the tissue present.

John C. da Costa, Jr.,[7] in a careful study of dorsal percussion in enlarged tracheobronchial glands, states that in 18 cases carefully studied he found the following four groups of physical signs:

1. Interscapular dullness, above the level of the inferior scapular angles, or over the first seven thoracic spinous processes. To this group belonged twelve or two-thirds of the cases.
2. Infrascapular dullness, below the level of the inferior scapular angles, or over one or more of the five lower spinous tips. To this group belonged two cases.
3. General thoracic hyperresonance over the entire thoracic segment. To this group belonged three cases.
4. Normal percussion signs in the entire thoracic segment. To this group belonged one case.

From this study it can be seen that sound alone is not the best criterion by which to judge of the presence or absence of enlarged mediastinal glands.

Auscultation.—Auscultation is of little value in diagnosis except in cases where the new tissue is of such a nature as to constrict a bronchus, in which case weakened breathing in the part affected may be detected, or where the enlargements stand in such relationship to the chest wall as to cause increased transmission of the breath sounds.

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[6] Mediastinal New Growth, Allbutt-Rolleston: System of Medicine, vol. v, 1909.

[7] Dorsal Percussion in Enlargements of the Tracheobronchial Glands, American Journal of the Medical Sciences, November, 1913.

CHAPTER XVIII.

TUBERCULIN TESTS IN DIAGNOSIS.

General Considerations.—Now that we have at our command several different methods of making the tuberculin test, and are gradually learning their limitations, we have arrived at a place where we can state with increased definiteness the value of these measures.

Years ago, when the diagnostic value of tuberculin was first learned, the subcutaneous was the only tuberculin test known. It was believed that a reaction to a dose of 10 mgs. or less of Koch's Old Tuberculin was positive evidence of the presence of clinical tuberculosis; and that a failure to react to 10 mgs. at least, if it were repeated once later, was a definite sign that tuberculosis was not present. At that time the frequency of tuberculous infections as we know it today was not known.

A great prejudice against the use of tuberculin as a diagnostic measure soon arose because of the severe reactions which attended it; so, the tuberculin test did not make great headway until other forms of employing it were discovered.

In 1907 von Pirquet announced the cutaneous test. This was soon followed by the announcements of Wolff-Eisner and Calmette, of the conjunctival test, by Moro of the percutaneous, and by Manteaux of the intradermal. While other forms have also been suggested; yet these are the ones which have been employed to the greatest advantage. They have helped greatly to popularize the use of tuberculin in diagnosis, although they have also added considerable confusion on account of the different opinions as to interpretation.

When the cutaneous test was applied generally to children it was found that a very large proportion of them reacted promptly. It shows that most children are already infected with tuberculosis by the time they reach their fifteenth year. This fact has caused great confusion and challenged the worth of all tuberculin reactions in adults, though I believe unjustly.

All tuberculin reactions, except the general reaction, are antigen-antibody or immunity reactions. The general reaction, on the other hand, is a toxic reaction as described more fully in Volume II, Chapter XL. Immunity reactions are an expression of the reaction which occurs between the specific ferments or antibodies which are called into being as a result of the stimulation of the body cells by the tuberculin introduced into the tissues. Whenever an inoculation, but particularly an infection with tubercle bacilli takes place, the cells of the body of that individual are so changed that they are sensitized against further attacks of the tubercle bacillus and its products; consequently the general opinion of the tuberculin reaction being evidence of active tuberculosis has gradually changed and in the minds of most clinicians the fact that an adult reacts to tuberculin has gradually lost its significance. This change in attitude toward the tuberculin tests is not wholly warranted; but our newer knowledge shows that reactions must be more carefully interpreted. While a tuberculin reaction will occur as long as the cells are sensitized to the products of the tubercle bacillus (as long as specific defense is present) and as long as they are active in producing specific antibodies against the tubercle bacillus, yet there is a recognized difference in the manner in which the body cells respond when fighting an active infection as compared with a quiescent or healed lesion. Observation has led me to believe that these reactions differ greatly and are of decided value. I have endeavored to interpret reactions, particularly the skin reaction, according to certain recognized principles in the establishment of immunity, and, while there are many times when it will not give the desired information, yet it will help as often as most measures in diagnostic use.

It is most reliable in persons whose health is not undermined, because a positive reaction depends on the body cells having their normal reacting capacity and being able to respond with specific cell sensitization and with the production of specific antibodies when brought in contact with the tubercle bacillus or substances derived from it. The body cells are most active in defense during the time when they are fighting active infection, and less active when this infection has become quiescent or healed. With these facts before us we are justified in believing that this difference in degree of specific cell sensitization (specific cellular defense) shows in the manner in which the cells react to tuberculin. Active tuberculosis, being accompanied by marked cell sensitization, responds quickly and markedly to the tuberculin test, while quiescent or healed lesions respond lightly and slowly. This I have found clinically. Those patients who are suffering from active tuberculosis, as diagnosed by other methods, respond, as a rule, to the skin test, promptly, reaching the maximum of their reaction within the first twenty-four to thirty-six hours; while those in whom the disease is quiescent or partially so, respond to a less degree and more slowly, the maximum reaction being reached somewhat later if it appears as more than a very slight reaction. In a patient with suspicious clinical history and a prompt maximum tuberculin reaction, I would be inclined to make a diagnosis of active tuberculosis, whether physical examination reveals the focus or not. If, on the other hand, I found a negative reaction under these circumstances, or only a slight reaction, and the patient's physical condition was such that I would expect a marked reaction in the presence of an active lesion, I would be inclined to feel that the patient was free from activity. A failure to react, or a slight reaction in one whose general condition is far below par, would not influence my opinion in the least.

One difficulty in making diagnoses is that we lose sight of the fact that tuberculosis is often concealed. Clinical symptoms of tuberculosis might be present and, yet, upon physical examination nothing be found that would make one suspicious of the lung being involved. The lesion might be in the mediastinal glands or any other glands or tissues of the body. I do not doubt that a great deal of our distrust and error in diagnosis of tuberculosis is due to this very fact. We are always expecting to find it as a pulmonary involvement, when it may be an involvement of any one of many organs. If we come to the point of recognizing tuberculosis as such no matter where found (and that is the way we should recognize it; at least we should recognize active tuberculosis as active tuberculosis whether it be in the bone, or the joint, or lung, or wherever found) then we would be making real genuine progress in diagnosis; but, as it is today, if we are not allowed to make a diagnosis of active tuberculosis unless it be in some portion of the body where it is going to seriously interfere with function, such as in the lungs, meninges, bones, joints, or peritoneum, and if we are to ignore it when in the hidden glands, simply because we cannot see the focus, then we are going to continue to be greatly confused in trying to harmonize physical findings, clinical history, and the various reactions. Syphilis is syphilis no matter where we find it, and a positive Wassermann demands treatment. This is recognized by all authorities whether the site of the lesion is found or not. Why consider syphilis so dangerous and ignore tuberculosis, or wait for it to manifest itself in some important organ before giving the patient the benefit of scientific treatment? The results of such a policy are shown by the death rate.

It is necessary to bear in mind in employing tuberculin in diagnosis that the reactive capacity of different tissues and different patients as well as that of different tuberculins and different preparations of the same tuberculin differs. Sometimes patients will not react to tuberculin even though tuberculosis is present. Such patients, as a rule, are in a low state of vitality and have built up no specific resistance to the tubercle bacillus; or, if they have, it is in abeyance at the time. This is particularly noticed in the use of the skin test in persons suffering from acute infectious diseases, such as diphtheria, whooping cough, scarlet fever, meningitis, and particularly measles; and it is true of all tests to some extent in persons suffering from fever and cachexia and those who are in a low state of vitality.

The subject of the relationship of hypersensitiveness to the character of the lesion has been discussed from the experimental standpoint by A. K. Krause.[1] In these experiments he shows that the nature of the reaction depends upon the activity of the disease. This is in accord with the observations which I have made clinically and discussed previously. I wish to quote his general conclusions based not only on his own work but that of Baldwin and Römer, which are as follows:

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[1] Experimental Studies on the Cutaneous Reaction to Tuberculo-Protein, Journal of Medical Research, New Series, vol. xxx, September, 1916.

1. "There is no cutaneous hypersensitiveness without a focus (tubercle).

2. "This hypersensitiveness appears coincident with the establishment of the focus.

3. "It diminishes with the healing of the focus.

4. "It varies directly with the intensity of the disease, which in its turn is dependent on the virulence of the invading bacillus."

The Subcutaneous Test.—Koch's Old Tuberculin is employed in making the subcutaneous test. While any tuberculin may be used, yet there are certain reasons why Koch's Old Tuberculin is best. The tubercle bacillus is made up, or is capable of being divided into many different substances which Much has characterized as partialantigens; for example, there are several different proteins, also several different fats that can be derived from it. The preparations of tuberculin are made in different ways and contain these various partialantigens in different proportions; in fact, in certain preparations some of these partialantigens are almost wholly wanting. The tissues of patients suffering from pulmonary tuberculosis contain antibodies to these different partialantigens in different proportions; in fact, sometimes antibodies to some of them are almost wholly wanting; consequently, in our choice of a tuberculin for diagnostic purposes we should employ one that contains the greatest number of partialantigens. Koch's Old Tuberculin fills this description best of all preparations, and consequently is the one we shall discuss. If several different forms of tuberculin are used at the same time in making the test, the reactions from the different preparations are not always uniform, as can be readily understood; but a reaction to any form of tuberculin is proof that antibodies to the particular partialantigens present in the preparation exist in the body tissues and such reaction is of diagnostic importance. It is further probable that, unless the patient has been inoculated with that particular partialantigen, the specific antibodies present are there, as a result of a tuberculous infection.

The dilution of tuberculin for the test is simple. Dilutions should be made fresh at the time of each dose; or at least at intervals not longer than ten days, because the weaker dilutions do not remain active indefinitely. The original solution, however, if tightly corked, may be kept for a long time without losing its strength.

Dilutions may be made by a graduated pipette or syringe. As a diluent, normal salt solution, plus two-fifths of one per cent carbolic acid is used. Two dilutions of the original tuberculin should be made. One a 1:100 is made by taking 0.1 c.c. of original solution and diluting it with 9.9 c.c. of the diluent. Of this solution 0.1 c.c. equals 1 milligram (0.001 c.c.) of tuberculin, and a syringeful or 1 c.c. equals 10 milligrams (0.01 c.c.) of tuberculin. To make the 1:1000 dilution, 0.1 c.c. of the 1:100 dilution should be taken and diluted with 0.9 c.c. diluent. Of this dilution, 0.1 c. c. equals, 0.1 milligram (0.0001 c.c) of tuberculin, and 1 c.c. equals 1 milligram (0.001 c.c.) of tuberculin. These two dilutions are sufficient, because from them we can conveniently measure the doses, ranging from 0.1 mgm. to 10 mgms., which are ordinarily used in making the test.

Temperature.—Preparatory to administering the subcutaneous test, a period of observation of the patient is essential. During this time, the patient should live as carefully as possible, saving himself from all unnecessary work and worry. It is best to have the patient confined to bed both during the period of observation and the time that the subcutaneous test is being made; but it is not essential, and cannot always be carried out, especially in clinic patients. It is essential, however, that the patient live under the same conditions during the period of observation and the making of the test. If he is in bed during the time of the test, he should be in bed during the period of observation. If he is to be up and around during the test, he should be the same during the period of observation.

It is also essential that a record of the temperature and pulse be kept during both the period of observation and during the period of making the test. This should be made accurately. The temperature should be recorded at least four times a day, preferably on awakening in the morning, at twelve, four and eight o'clock. The thermometer should be held in the mouth sufficiently long to guarantee full registration. This should be at least five minutes; and if the weather is cold and the patient is surrounded by cold atmosphere, it should be longer because it will often take ten minutes or more to warm the mouth so as to secure full registration. During the time that the patient is holding the thermometer he should not talk, because opening and closing the mouth interferes with accurate registration.

It is best to administer the tuberculin at night so as to have the opportunity of observing any rise in temperature that would follow during the succeeding day. The temperature reaction usually begins sometime between eight and sixteen hours after the injection, so the rise in temperature is usually found during the succeeding day if the test is given at night. The reaction time, however, varies greatly under different circumstances.

The subcutaneous test is applicable to any patient who is in an afebrile condition or any patient who is running a slight degree of temperature. As a rule, if the temperature is above 99.6° or 100°, it is not applicable. In fact, in a patient who is having a daily maximum of 99° to 100°, the diagnosis, if due to tuberculosis, should usually be made upon clinical history and physical examination if the disease is in the pulmonary tissue or some other part of the body that is accessible. If not, some other form of the test should be used. If temperatures above that mentioned are due to tuberculosis it means that there is a considerable degree of activity present; and, if marked activity is present, a tuberculin reaction such as that which is apt to follow the injection of tuberculin for the test, is best avoided; although I can hardly conceive of a single tuberculin reaction producing any serious trouble.

It has also been said that this form of test is not particularly applicable in hysteria because the patient may become excited and suffer a rise of temperature from the nervous state, which might be mistaken for the reaction. If the peculiar characteristics of the tuberculin reaction, however, are remembered, it will be seen that there is a difference between the rises of temperature from tuberculosis and those due to other transitory conditions. It will further be seen that any rise of temperature due to a tuberculin reaction is usually accompanied by the chain of symptoms which make up the general reaction.

In suitable cases for the test, that is, in slight infiltration without much temperature, no matter where the lesion is located, except in the meninges, the subcutaneous test is practically without danger.

The injection may be given in any portion of the body. For convenience, however, I am inclined to use the forearm, upper arm, or sometimes the back.

The question of dosage is an important one although it might not seem so from the variance in methods in vogue. There is much diversity in methods of administering the subcutaneous test. Koch suggested three doses, 1, 5, and 10 milligrams, the latter to be repeated in case of failure to react. These to be given with two or three days intervening between doses. The writer's method of giving tuberculin in diagnosis is as follows: The dosage is gauged according to the earliness of the lesion, the tissue involved; the degree of activity suspected; and the physical condition of the patient. If the lesion is early and inclined to be active, smaller doses should be used than in case it is more extensive or the lesion is fairly quiescent. If the patient is reduced in vitality, smaller doses are employed than if the patient is stronger. In a child the dose is reduced from one-tenth to one-half that given to an adult. For the average adult my initial dose is one milligram. Two days later, if there is no reaction, I give 3 or 5 milligrams; three days later, in case of no reaction, I give 7 or 10 milligrams, according to the conditions. If the patient is a child, or one whose vitality is lowered, I would generally begin with one-tenth, then 1, 3, and 5 milligrams. While a patient might react to 10 who does not react to 5, for the sake of safety higher doses are rarely employed. Nearly all patients suffering from early active tuberculosis will react to 10 milligrams if the proper increase and spacing of doses is followed.

A positive reaction to a subcutaneous injection of tuberculin may show in three ways, by local, focal, or general phenomena. The local reaction consists in an irritation at the point of injection. While this, of itself, is not generally considered as a positive reaction in this form of the test, in reality it is as much so as any other local reaction.

The focal reaction is a reaction in the tuberculous focus resulting from the combination of substances found in or elaborated from the tuberculin with the sensitized cells about the tuberculous focus; or as a result of specific antibodies, whose formation is stimulated by the introduction of tuberculin, combining with the products of the tubercle bacillus found in the focus of infection. The phenomena which appear are such as accompany any non-specific inflammation. If the reaction is light, a slight hyperemia only occurs, but if it is severe, the signs of a more serious congestion with exudation appear, accompanied by an increase of the usual signs and symptoms which previously existed.

If the focal reaction is in a pulmonary focus it may be accompanied by a cough even when one had not existed previously; or an increase if it had previously existed. An exaggeration of the signs on auscultation may also take place, and fine râles may be increased or brought out where they had not previously been detected. The detection of a focal reaction in the lung, however, is not an easy matter, and is of little use to anyone but an expert stethoscopist. In visible tuberculous ulcers, such as those in the larynx, the focal reaction may be seen as an increased hyperemia; in glands it sometimes shows as an increase in the size, with pain, swelling and redness; in the kidney, pain, increased bacilluria, or hematuria may occur rarely, but must not be expected. In fact, the focal reaction, while giving more positive information, is, in the main, misleading and disappointing. It does not always occur following the doses administered.

A general reaction is accompanied by the same toxic symptoms as are noted when activity is present in a tuberculous focus, and these symptoms differ in degree the same as they do in the clinical disease. There may be a slight drowsiness, a feeling of heaviness, a tired feeling, or an aching, either slight or amounting to extreme discomfort, such as accompanies acute infection (tonsillitis or la grippe). The patient experiences an increased nervousness, sometimes insomnia. When the reaction is severe, a chill and even vomiting may precede a rise of temperature.

The temperature rise was formerly made the sole means of judging the subcutaneous test, and an elevation of two degrees was demanded for a positive reaction. This is no longer necessary, for those who have had experience have been able to demonstrate that the chain of symptoms previously mentioned almost always manifest themselves with a dose short of that which causes the two degree rise in temperature. The lighter symptoms also mentioned will sometimes appear without a rise in temperature, or with a rise of only a fraction of a degree and are just as specific. The effect of intercurrent troubles in producing similar symptoms must be considered, should any such arise.

The diagnostic value of the general reaction, which is more properly called the toxic reaction, depends upon the presence of specific proteolytic enzymes. When a small quantity of tubercle protein is injected parenterally into the body of a non-tuberculous subject, no recognizable signs of reaction follow, because the process required for its destruction is a slow one, and the toxic molecules are set free very slowly and in such small quantities that they are rendered harmless or excreted without producing recognizable effects. When injected into an individual who is infected with tuberculosis, on the other hand, specific proteolytic enzymes capable of splitting it up into less complex molecules, are already present, and act upon tuberculin as soon as they come in contact with it, setting free the toxic molecules rapidly and in large quantities. The character of the reaction depends upon the rapidity with which the toxic molecules are liberated; and this, in turn, depends upon the amount and activity of specific enzymes present. Inasmuch as these are present in greatest amounts, and most active, when the body is fighting an active infection, active tuberculosis, all other conditions being equal, reacts to smaller doses and more promptly than quiescent and healed tuberculosis. This gives the reaction its diagnostic value.

The general reaction manifests itself as the syndrome of toxemia, which acts through stimulation of the cells of the central nervous system and is expressed chiefly as a general sympathetic stimulation. The cause of the fever, as described in Volume II, Chapter XXX, is not so much the increase in heat production as it is a stimulation of the vasoconstrictors which interfere with the elimination of heat from the surface of the body.

The temperature following tuberculin has certain peculiarities which are almost diagnostic of themselves. If Koch's Old Tuberculin is employed, and it is best, the temperature usually comes on sometime between eight and twenty-four hours after the injection. The temperature usually rises gradually. It may take twenty-four hours to reach its maximum and then it falls gradually. I have never seen a reaction of more than a small fraction of a degree, which was positively due to tuberculin, in which the maximum temperature was followed in a few hours by a normal. The morning temperature on the day following a positive tuberculin reaction is nearly always above the usual morning register. The accompanying curve, from the author's monograph on tuberculin,[2] illustrates this point (Fig. 98).

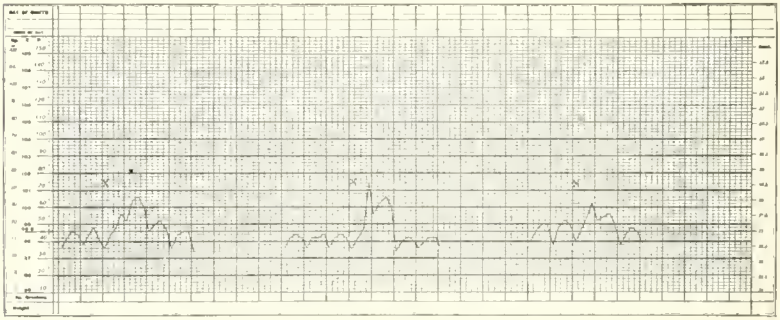

Fig. 98.—Three different temperature curves, illustrating tuberculin reactions. It will be noted that the temperature does not return to normal on the second day of the reaction. This is characteristic of the curve of the tuberculin reactions.

The Cutaneous Tuberculin Test (von Pirquet).—The cutaneous tuberculin test, which is commonly called the von Pirquet test, was brought forth by von Pirquet in 1907. He explains it as being a reaction of hypersensibility and due to a condition which he has described as allergy, from *ergeia*, meaning reactivity and *allos*, meaning altered. He means by this that in tuberculous patients the reactivity of the cells is altered from what it is in those who are non-tuberculous, and that this alteration manifests itself in a specific method of reacting when tubercle bacilli or the products of tubercle bacilli are brought in contact with them.

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[2] Tuberculin in Diagnosis and Treatment. C. V. Mosby Co., 1913.

The cutaneous, like the subcutaneous test, depends for its diagnostic value upon the fact that the infected individual possesses within his body specific proteolytic enzymes which rapidly split the protein molecule contained in the tuberculin into simpler molecules. While the subcutaneous reaction depends on the toxic molecule, the cutaneous, along with the percutaneous, intradermal and conjunctival, depends upon the sensitizing molecule for its reactive phenomena.

This reaction is more specific than the toxic reaction; in fact, the factor which makes the toxic reaction (subcutaneous) of any value at all in diagnosis, is the rapidity of the destruction of the tubercle protein, resulting in prompt liberation of the toxic molecule.

The real diagnostic worth of the cutaneous test is not in the mere presence or absence of a reaction. Some evidence of sensitization should be expected in all patients who have harbored infection within their bodies. Clinical experience, however, seems to show that this gradually lessens until it may finally disappear after the infection has been healed for a term of years. The nature of the reaction, however, affords valuable diagnostic evidence. It gives an idea of the degree of sensitization present. If marked sensitization is present it suggests that the patient is, or has been recently, fighting active tuberculosis. Otherwise there would be no need for the presence of the large amounts of specific enzymes which are responsible for the reaction.

I have gradually learned to suspect that the patient who reacts promptly with a marked reaction reaching the maximum within the first twenty-four or thirty-six hours, does so because the body is well supplied with specific enzymes as a result of an unhealed focus. The degree of promptness of the reaction in this test, the same as in the preceding, depends upon the rapidity with which the tubercle protein is split, consequently, upon the number of specific enzymes, which are governed by the urgency of defense.

Before making the cutaneous test, the skin should be cleansed with soap and water and allowed to dry. The test is then made by placing a drop of the solution of tuberculin to be used, on the skin and producing an abrasion of the skin through the drop. This method is much better than to make the abrasion first and put on the tuberculin afterwards; because, by this method of making the abrasion, the tuberculin is mechanically pressed into the tissues and thus comes in surer contact with them. The scarifier devised by von Pirquet is much better than the scalpel because it is desirable not only to determine whether or not a reaction is present, but the degree of the reaction. This cannot be so accurately done when there has been considerable trauma, as often occurs when the scalpel is used. It is well to make a control abrasion without tuberculin, for the purpose of comparing the traumatic reaction with the tuberculin reaction if present. In making this test, I, personally, prefer to use 100 per cent strength of tuberculin, although 25 per cent and 50 per cent may be used, if desired. I have never seen any harm from the 100 per cent and have found some positive reactions with the full strength which were more or less doubtful with weaker dilutions. Tests may be made anywhere on the body; but, for convenience, I prefer the forearm, although there are some reasons in favor of choosing a portion of the skin which is more delicate, such as that over the thorax. Sometimes the delicate skin will give a more marked reaction than the skin of those parts which are less sensitive.

This test should be given in a definite manner. Its very simplicity often leads to a haphazard method of making it and this militates against its usefulness. The tuberculin should be brought into close contact with the subcutaneous tissue and should be allowed to remain in contact for a sufficient time for absorption to take place. Five minutes is usually sufficient. The patient should then be instructed not to allow the clothing to wipe it off. Some clinicians cover the abrasion with a gauze protection, but this is not necessary. Not only the reaction, but the strength of the reaction, and the time of its appearance should be noted.

A positive reaction consists in an area of redness appearing at the seat of the inoculation. Sometimes it is only a slight redness of the tissue surrounding the trauma, which can hardly be

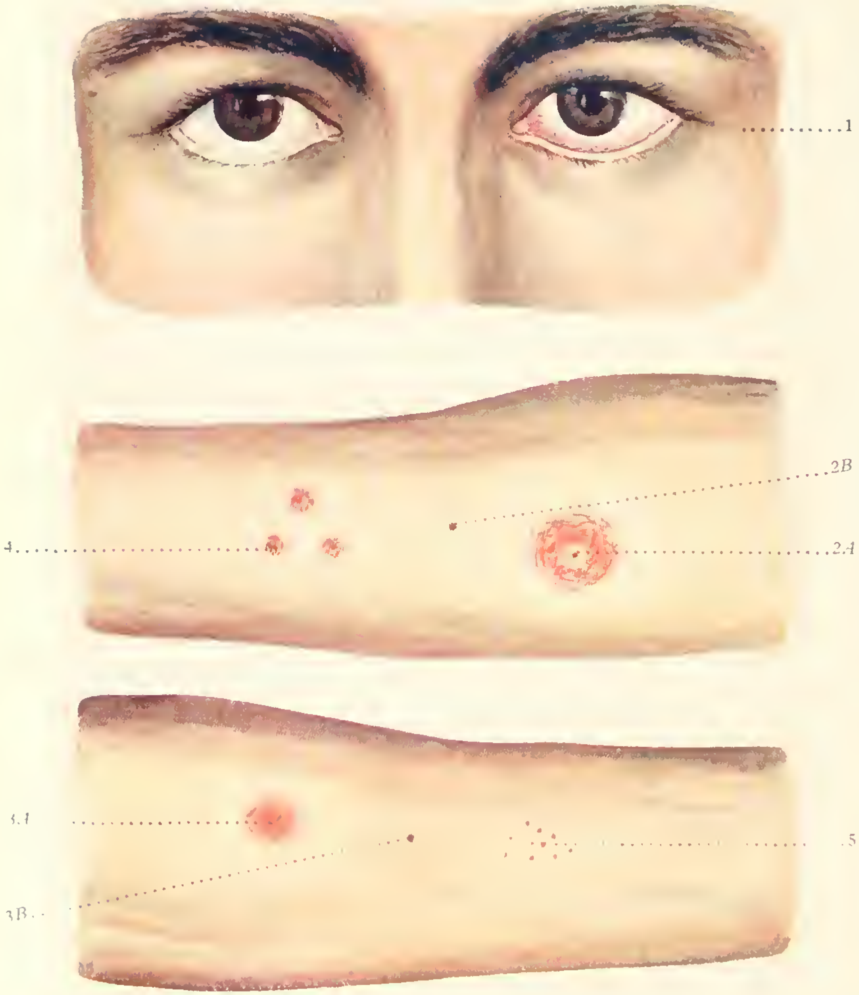

PLATE VI

1. Conjunctival reaction of left eye.
2*A*. Von Pirquet reaction, well marked; *B*, control site.
3*A*. Von Pirquet reaction, moderate; *B*, control site.
4. Moro reaction, well marked.
5. Moro reaction, slight.

differentiated from the traumatic reaction of the tissues. Sometimes it consists of a papule which may even form a vesicle. Surrounding the papule there is usually an area of redness of varying extent, and at times the reaction can be traced out along the lymph channels for a distance of several centimeters. A very marked reaction may be four or five centimeters in diameter. It is possible that sufficient tuberculin may be absorbed to cause a temperature reaction in very sensitive individuals. Such instances have been reported but the writer has never seen it occur. Plate VI (3 *a* and *b*) illustrates the cutaneous reaction.

The Percutaneous Test (Moro).—The percutaneous test is made by rubbing a mixture of equal parts of Koch's Old Tuberculin and lanolin into the uninjured skin. Of this an amount equal to the size of a pea is used. It is best to use some portion of the skin that is protected and sensitive. This test is not as reliable as the cutaneous because of more or less error in securing absorption. The reaction usually occurs in twenty-four to forty-eight hours after the inoculation and shows as a number of small red spots, but if the reaction is severe they may take the form of small nodules lasting for several days. (See Plate VI, 4 and 5.)

The Conjunctival Test (Wolff-Eisner).—The conjunctival test which was brought out by Wolff-Eisner and later by Calmette is gradually falling into disuse, owing to the more simple cutaneous and intradermal methods.

Koch's Old Tuberculin is used for the test. Two dilutions are made, a one and a two per cent strength. One or two drops of the one per cent is instilled into one eye. If no reaction occurs, later a two per cent should be instilled into the other eye. A positive reaction is shown by the development of hyperemia. If the reaction is slight there is only a faint redness of the conjunctiva, but there may be a marked conjunctivitis, even purulent in character, if the reaction is severe. It should not be used in any case where the eye is diseased, particularly if the disease is of a tuberculous nature. This reaction is shown in Plate VI, 1.

The Intradermal Test is made by injecting a few drops of a dilution of 1-1000 or 1-5000 of Koch's Old Tuberculin between the layers of the skin. A positive reaction shows as an induration.

CHAPTER XIX.

THE X-RAY AS AN AID TO THE DIAGNOSIS OF PULMONARY TUBERCULOSIS.

Relative Value of Physical and X-Ray Examinations.—The difficulty as well as the importance of early diagnosis of tuberculosis reveals itself in the great number of methods which have been suggested to detect this disease during the period when the pathological changes are slight. The most important methods for the diagnosis of early clinical tuberculosis are the clinical history and physical examination of the patient. These two alone, if the history is taken with sufficient care and the examination is made with sufficient skill, will determine the presence or absence of tuberculosis in nearly all instances.

As supplementary aids to these important measures, however, we have the examination of the sputum for bacilli, lymphocytes, and albumin; the complement deviation and opsonic tests; the tuberculin tests; and the x-ray. Each of these methods of examination may become an important aid under certain circumstances. This is particularly true of the examination for bacilli, the tuberculin tests and the x-ray. These supplementary tests, however, should be considered only as aids to diagnosis and should be used in conjunction with the clinical history and the physical examination.

When the x-ray was first discovered it was hoped that a method had come into vogue which would give an accurate picture of all conditions within the thorax, but it was soon learned that such was not the case.

It has taken a number of years to determine the relative value of the x-ray examination as compared with the clinical examination. Clinicians who have had most experience and are best able to judge are in accord in their opinion that an accurate clinical history and a skillful physical examination will reveal changes which result from tuberculosis earlier than can be shown by the x-ray examination. From the practical standpoint, however, this is not the question of greatest importance. The question is: how may the x-ray be used to give the greatest aid in the diagnosis of early tuberculosis? While the value of a skillful clinical examination cannot be questioned, yet it is but fair to admit that proper accuracy and skill are not always applied.

Cases of early tuberculosis will be found among every man's patients; and every man who does any considerable practice will at times suspect tuberculosis. Many will overlook the disease because of their inability to make the clinical examination with sufficient skill. Diagnosis at such times will be facilitated either by a skillful clinical consultant or by having the aid of an expert radiographer. Even the men who make the most careful examinations find themselves puzzled now and then, and can be aided greatly by the study of an x-ray picture, particularly the stereoscopic picture made by one skilled in pulmonary röntgenology. It is important in this connection, to have in mind that the plate and screen are, at least equally and probably more likely to present inaccuracies than the careful physical examination. It must not be taken as an infallible picture, nor must its interpretation be taken as being without error.

The practical question which confronts us in this discussion is: can a more general use of the x-ray result in causing diagnoses of pulmonary tuberculosis to be made earlier than they are being made by present clinical methods? The answer to this question cannot be given except by considering the relative skill of the examiners. It seems to me that whenever the skill of the examiners is at all nearly equal, preference should be given to the personal clinical examination rather than to the impersonal x-ray plate. The human side of medicine must not be underestimated. The analysis of the case, the symptoms, the clinical course, and the data found on physical examination as belonging to and being a part of a process within a human being is all important. In other words, the ultimate diagnosis should be made by the clinician. However, the employment of an expert x-ray operator, the same as an expert clinical consultant by men who are not able, or at least by those who do not make careful chest examinations, would undoubtedly reduce the percentage of error in diagnosis very materially. On the other hand, reliance placed on poor plates made by unskilled workers can only mislead. Such x-ray examinations are on a par with poor physical examinations, and cannot be relied upon.

There has been considerable discussion as to whether or not active tuberculosis can be differentiated from healed tuberculosis by the x-ray picture, particularly when the lesion is young. Skilled operators who use up-to-date technic and who have most experience in chest work claim it possible to do so. Such diagnoses, however, should not be accepted without careful comparison with the data found upon clinical examination. The earliest pathological changes in tuberculosis of the lung are not gross. There is no calcified tubercle, neither is there fibrous tissue. There are fresh tubercles surrounded by areas of exudative inflammation of a mild degree. This condition is rarely diagnosed except by expert clinicians, and it is decidedly questionable whether a shadow would be shown on the screen as a result of it. The fluoroscope should show the altered motion of the diaphragm; and this, together with the changes in the tone (spasm) of the apical muscles should direct the examiner to the pulmonary involvement.

Minor[1] says: "After having used the röntgen ray fluoroscopically in all his examinations for the past seven years, the writer believes that in the majority of cases an expert physical diagnostician will be able to make a diagnosis of incipient tuberculosis sooner than will the radiologist; but, in a few cases, the latter will discover small foci of tubercle in the lung which neither auscultation nor percussion would reveal. There are certain pulmonary conditions, especially enlargements of the tracheobronchial glands and peribronchial infiltrations which can be diagnosed far earlier and better by this method than by any other."

Gregory Cole[2] states that the x-ray plate, when properly interpreted, will make the diagnosis of early pulmonary tuberculosis earlier than either the fluoroscope or physical examinations.

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[1] Klebs: Tuberculosis, D. Appleton & Co., 1909.

[2] The Radiographic Diagnosis and Classification of Early Pulmonary Tuberculosis, American Journal of Medical Sciences, July, 1910.

Vincent Y. Bowditch[3] says: "In spite of the great advance that has been made in the practical use of the x-ray of late years, I can but feel that we have not yet sufficiently studied the causes of the phenomena shown by this method to warrant our drawing what have seemed to me at times hasty conclusions as to the amount and nature of disease which may be present in the lung. One thing is absolutely certain in my mind, viz.: that I have very little faith in the findings of anyone but those who are experts in this department of science, but I must unhesitatingly condemn the diagnosis of those who, upon the insufficient ground of their own supposed findings in the use of the x-ray, give their patients unnecessary alarm and anxiety as to their bodily condition."

Bonney[4] made a careful comparison of physical examinations and radiography and states: "As a result of this inquiry, however, previous convictions as to the slight practical value of the x-ray in the diagnosis of *very incipient* cases without well defined structural lesions have been substantially confirmed."

Heise and Sampson,[5] after an analysis of their own experience in ninety-five very early and difficult cases, take a very commendable stand on the comparative advantage of physical examination and the x-ray, as follows:

"1. When the history and physical examination were positive, the x-ray showed a lesion in 77 per cent and failed to show a lesion in 23 per cent.

"2. When the history and physical examination were negative, the x-ray failed to show a lesion in 58 per cent and disclosed one in 42 per cent.

"3. When the history was positive and the physical examination negative, the x-ray showed a lesion in 60 per cent and failed to show one in 40 per cent.

"4. When the history was negative but the physical examination was positive, the x-ray showed a lesion in 50 per cent and failed to show one in 50 per cent.

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[3] What the General Practitioner Should Know About Incipient Pulmonary Tuberculosis. Boston Medical and Surgical Journal, November 25, 1915.

[4] Pulmonary Tuberculosis and Its Complications. W. B. Saunders Co., 1908.

[5] The X-Ray in the Diagnosis of Pulmonary Tuberculosis. Interstate Medical Journal, vol. xxii, No. 10, 1915.

"At first thought these results may seem to indicate that the x-ray has but little value as an aid in diagnosis. But when we consider that the basis of classification and the class of cases in which the comparison was used, the advantage that the x-ray affords is really obvious. In only 23 per cent of all were the history and physical examination both positive. In 35 per cent of the cases both history and physical examination were doubtful or negative. The history only was positive in 32 per cent and the physical examination alone was positive in 10 per cent. As the disease is more advanced naturally the percentage of definite lesions seen in the plate becomes proportionately greater. And it is a fairly common experience to find more extensive involvement upon x-ray examination than is detected by physical signs. At times the difference is so great as to be astounding.

"Plates taken at intervals in the course of the disease will show marked changes in some instances and at times these changes are detected before physical signs have perceptibly altered.

"In the differential diagnosis of mediastinal and lung conditions the value of the x-ray is unquestionable, but this phase of the subject will not be dealt with here.

"In conclusion, we would like to state that in our opinion the x-ray in diagnosis can be made to give, as nearly as possible without exploration or autopsy, evidences of the existing pathology in the lungs. It has been of distinct advantage in the diagnosis and better understanding of existing lesions in the lung, and with plates taken at intervals, changes for better or worse have been noted in the areas involved. Owing, however, to the fact that the x-ray is a shadow projector only, we do not think it infallible and that it should at the present time, at least, replace all other methods of diagnosis. Rather do we think it should be used in cooperation with all methods—as an aid rather than an infallible means."

In using the x-ray it must be remembered that slight lesions produced by early tuberculosis may not produce sufficient changes in the density of the tissues to alter the shadow, and yet may be detected by expert physical examination.

There are several portions of the lung which should be examined especially carefully for tuberculosis, although foci may appear in any part. Thanks to the x-ray we have learned of the frequency of hilus infection. We have found it necessary to change our ideas of physical diagnosis and search for early tuberculosis in wider areas than formerly. While the apex still holds its place as the common seat of early clinical tuberculosis, yet we commonly find infections near the hilus, running out into the tissue in all directions, but particularly toward the apex and the base. These hilus infections are especially difficult of detection on percussion and auscultation and here the x-ray has given great aid.

Interpretation of Plate.—It requires experience to read and interpret x-ray plates. While there is room for much error in making the plate, one must not lose sight of the fact that the greatest skill and judgment is required to interpret the plate.

We should expect the dense fibrous tissue of the healed tubercle and the calcified tubercle to interfere most with the penetration of the rays, consequently we would expect the shadows cast by such structures to be more definite and clear cut than those produced by tissue of lesser density. On the other hand, we would expect active tubercles, which are surrounded by an exudative inflammation, whether isolated or conglomerate, to produce a hazy undefined shadow, and the latter particularly to show a mottling of the plate.

The shadows cast depend on a great many things such as the character of the tube, the exposure, the depth of the lesion, and the chemical constituents of the focus. Assmann[6] made a careful study and comparison of x-ray findings and postmortem findings. He says: "At times the x-ray shows details of unexpected minuteness: for example, nodules scarcely as large as pin heads are at times shown, exact in position, size and relationship. Again, nodules as large as walnuts fail to appear on the plate and areas of thickened tissue even larger than this, cast no shadows or shadows which are scarcely recognizable. Aside from the thickness and specific chemical composition of the focus, the detail of the shadow which it produces depends upon its depth in the tissues."

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[6] Erfahrungen über die Röntgenuntersuchnug der Lungen, Jena, 1914.

Normal Hilus and Trunk Shadows.—The normal hilus shadow and the normal trunks running out from the hilus must be understood in order to correctly interpret the abnormal. Unfortunately, the interpretation of these shadows is accompanied by great uncertainty. These shadows differ with age, being slighter in children and becoming more pronounced as the individual grows older. As Dunham[7] well says: "Thus, in children under twelve . . . the trunks and marking can only be made out on plates which are taken very rapidly, and in subjects over sixty the changes are so marked that they would indicate tuberculosis in a youth of eighteen."

There has been a great deal of speculation as to the cause of the trunk shadows in the x-ray plate of the normal lung. If this could be definitely determined it would simplify the reading of the plate in pathological lungs. Some (De la Camp, Arnsperger) believe it is the bronchi; others (Albers Schönberg, Holzknecht) the blood vessels; while still others think it is a combination of the bronchi and blood vessels.

Assmann has thrown light on this point experimentally. He tied the vessels of one lung of a dog before killing, leaving the vessels of the other free. He then removed the lungs, washed the blood from the free side, inflated the lungs, and made plates for study. The resulting picture showed "thick solid branched trunk shadows on the side filled with blood, which were absent on the other side." In discussing the experiment he says: "Therefore it is made clear that the elastic vessels filled with blood cause intensive shadow markings in the form of well defined branched trunks." The author brings forth further evidence on this point in pointing out that the hyperemic lung shows enormously large trunk shadows in comparison with the normal lung. He further quotes Weber and Owen who filled the bronchi with shot so loosely that the individual shot were separated from each other. The shadow of the shot was found in a clear background instead of in the shadow trunks of the plate.

Since all structures which interfere with the passage of the rays, increase the shadows relatively on the plate, it seems to me that we must assume that blood vessels, lymphatics, and bronchi all have a part in causing the shadow markings of the normal lung; and that thickening of the walls of the bronchi and lymphatic trunks, or dilatation of the blood vessels would cause increase in the shadow and should be considered as pathological. This increase in trunk markings is to be separated from the shadows cast by the pathological changes in the bronchi and pulmonary tissue itself.

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[7] Stereorœntgenography. Pulmonary Tuberculosis. The Southworth Co., 1915.

In considering the effect of the blood vessels in the production of the shadows, we must bear in mind that the contrast between the solid blood column and the light air containing lung tissue is exceedingly great.

Stürtz[8] suggested that the markings in incipient tuberculosis extended from the hilus to the apex and that the apex was infected through direct lymphatic extension. Such a condition seems extremely unlikely upon pathological grounds. While bacilli may make headway against the lymph stream (Tendeloo) yet it is difficult to believe that they could bridge the gap between the hilus and the periphery of the lung without the intervening tissue becoming invaded.

Dunham[9] has described these same markings extending from the hilus to the periphery; but insists on certain special characteristics as being indicative of tuberculosis. His studies were made with the stereoscopic plate. To quote from his own writings:[10] "The anatomical explanation of this altered shadow picture, I am not at this time ready to discuss, though I believe it is due to connective tissue changes. I am sure, however, that the increase in the hilus shadow, the thickening of the trunks, together with such alterations in the linear markings as increase in density and breadth, studding, interweaving and extension to the periphery, constitute a shadow picture characteristic of early pulmonary tuberculosis.

"As the lesion progresses the alterations become more marked, the linear markings more broken up and irregular, the studdings become larger and denser, the interweavings closer until eventually all the markings are blotted out and the whole area appears as a unified mass of increased density, due to the presence of gross areas of consolidation within the lung."

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[8] Die lymphangitische Entstellung des Lungenspitzencatarrhs von den Hilusdrusen aus 4 Versammlung d. Tuberkulose-Ärzte, Berlin, 1907.

[9] Johns Hopkins Hospital Bulletin, vol. xxii, No. 245, July, 1911; also Stereorœntgenology, The Southworth Co., 1915.

[10] Stereorœntgenology, The Southworth Co., 1915.

Until the cause of these changes in the linear markings is definitely settled we can only speculate as to the manner of their production. It seems to me, however, that the key to the explanation lies in: (1), The cause of the linear markings; and (2), the fact that drainage is toward the hilus.

If the normal trunk shadows, both large and small, are due to a combination of blood vessels, bronchi and lymphatics, let us inquire into what there is in early tuberculosis which could enlarge these and make them cast a broader and denser shadow.

We could not expect thickening of the larger bronchi to take place this early. We might expect a certain amount of obstruction in the circulation to be caused by the infiltration, for there is some hyperemia associated with these early infiltrations, although the amount is much less than that which occurs later in the disease. It is also not at all improbable that as the products of inflammation are carried through the lymphatics away from the diseased areas toward the hilus they irritate and produce changes in the tissues through which they pass, which might cause slight alterations in the shadow. These two factors seem to me to be worthy of consideration in accounting for changes in the shadow markings. The distension of the blood vessels is especially important in connection with the studies of Assmann quoted above.

Methods of Using the X-Ray in Pulmonary Diagnosis.—The x-ray may be employed in three different ways in the examination of the chest: First, in making the single plate; second, the stereoscopic plate; and third, in making a fluoroscopic examination.

Each method has merit. Wherever stereoscopic plates can be had they have a great advantage over the single plate in that the different structures stand out in their true relationship. Plates are valuable as permanent records of conditions. The fluoroscope, however, is more flexible, but fails in definition. It permits of studying the chest from different angles. It also offers an advantage in studying the motility of the chest wall and particularly the diaphragm. The interference with the action of the diaphragm in early tuberculosis, as pointed out by Williams in 1907, is probably due to two factors: the reflex disturbance in motility produced through the phrenics by irritation of the nerve cells in the third, fourth, and fifth cervical segments of the cord by afferent impulses which travel through the sympathetics and rami communicantes from the inflammation in the lung as first suggested by the writer; and a diminished elasticity of pulmonary tissue caused by the infiltration in the lung as suggested by Hofbauer and Holzknecht. The fact that the diaphragm phenomenon is found very early, before there is sufficient disturbance in the elasticity of the tissues to cause it shows that some other explanation must be adduced for it in the very early lesion. Since other muscles taking their origin from the same segments as the diaphragm are involved equally early in the motor reflex, this suggests itself as the plausible explanation.

Some operators fail to confirm William's sign. This is probably partly due to a failure to understand the conditions surrounding its production. Clinically, the diminished motion of the chest wall may also be determined. Many observers have difficulty in detecting it, probably because of a failure to understand the normal and because of wrong technic. Careful, painstaking inspection and palpation of the bases by one who has studied the normal respiratory movements of these portions of the thoracic wall will rarely fail to detect departures from the normal. If the motions of the chest wall are studied both on normal and forced inspiration some departures from the normal will be found universally when the pulmonary tissue is the seat of active inflammation. To examine this portion of the chest either by inspection and palpation or by the fluoroscope, there should be no constriction of the waist to interfere with the normal movements of the parts; and the patient should be examined on both normal and forced respiratory effort. Sometimes deficient motion will show on normal respiration and not on deep respiration and sometimes the reverse is true.

In discussing the interference with the motility of the diaphragm it is well to call attention to the fact that diminished descent of the diaphragm may be due to pleural adhesions and certain abdominal conditions interfering with its movements.

It is not necessary to say that a diagnosis should not be made on the disturbance in function of the diaphragm alone. This phenomenon when found, however, calls for careful examination of the pulmonary tissue in order to discover its cause. The rationale of its production is made plain by the fact that the central tendon is supplied by the phrenics innervated by the third, fourth, and fifth cervical segments.

The stereoscopic plate is undoubtedly the greatest advance that has been made in the x-ray of the chest and I do not doubt that careful comparative examinations made by expert clinical diagnosticians and expert x-ray operators will, in the near future, be able to put the diagnosis of early pulmonary tuberculosis on a much surer basis than it has hitherto enjoyed.

Cases Illustrating Comparative Results of Clinical and Stereoscopic Examination.—The comparative results of clinical examinations and stereoscopic plates may be illustrated in the following cases. The writer analyzed the cases from the clinical standpoint, taking into account:

1. Clinical history and symptomatology.
2. Motor (spasm) and trophic (degeneration) disturbances in the neck and chest muscles including the diaphragm (diminished motion) and trophic changes (degeneration) in the skin and subcutaneous tissue over the chest wall.
3. Changes on palpation, percussion, and auscultation.
4. The results of the tuberculin test.

The stereoscopic plates were taken and read by Doctor Albert Soiland of Los Angeles. The diagnoses were made independently and then compared.

A comparison of the clinical findings with the x-ray findings in the cases will show a fair degree of agreement.

Case 2786.

Female, age 39 years; family history, negative.

Clinical History.—Not strong as a young girl. Better the last four years except for a severe attack of tonsillitis last year, for which the tonsils were removed. Three months ago began to cough and developed enlargement of the left anterior cervical glands. Has also had stiffness and pain in the left knee for one year, the exact nature of which is not known.

She has the following symptoms.

I. Toxic Group.	II. Reflex Group.	III. Tuberculous Process *per se.*
Malaise	Slight hoarseness for several months	Bronchitis for three months
Lack of endurance	Tickling in larynx at times very annoying	
Slightly disturbed appetite	Cough	
Loss of 3 pounds in weight	Pain in left shoulder	
Temperature 99.8		

Physical Examination.—Left pupil larger than right. Left sternocleidomastoideus, scaleni, trapezius, levator anguli scapulæ slightly degenerated, with well pronounced spasm. Left side lags. Percussion note slightly impaired over left apex.

Auscultation shows inspiration slightly roughened, with expiration slightly prolonged over left apex anteriorly and posteriorly.

Tuberculin Test.—Von Pirquet test (Koch's Old Tuberculin full strength) positive, reaching maximum of 1 cm. in 24 hours.

Diagnosis.—Active tuberculosis of the left apex.

X-Ray Findings.—

Left Side.—Apex slightly cloudy.

Several caseated areas in hilus, with generalized inflammatory deposit near same.

Lower lobe shows thickened walls of an old healed cavity.

Right Side.—Apex clear.

Hilus shows a moderate degree of fibrosis and calcified glands.

Case 2643.

A. P., female, age 6½ years.

Clinical History.—Patient suffered from recurrent attacks of cold during second and third years of life, for which adenoids were removed when eighteen months old, and again when two and a half years old. Patient was free from colds for one year. When four years of age had whooping cough which was mild. About three years ago patient took a cold which resulted in a severe bronchitis. Patient had considerable temperature and made a very slow recovery. It is questionable whether there was a pneumonic process present at the time. Following this attack the patient suffered from bronchitis for two months or more, for which she was taken to a mild climate for relief. From that time until the present, the patient has had repeated attacks of colds, which always ended in bronchitis; and from which recovery was slow. At the present time the patient is suffering from one of these attacks of bronchitis, and is brought for examination to determine the cause of same.

Physical Examination.—Inspection and palpation reveal the following: A fairly well developed child with the exception of the upper portion of the chest, which is decidedly flattened, being more markedly so on the right than on the left. Not only is the chest flattened, but there is some degeneration of the soft tissues on the right. There is no increased tone (spasm) of the muscles. Posteriorly the soft tissues are also slightly degenerated over the chest, but not markedly so. The *total density* of the chest as revealed by palpation is increased over the entire right lung, which shows most markedly posteriorly.

Percussion also shows a slight increase in the resistance to the finger over the right lower lobe posteriorly and the upper lobe anteriorly, the changes being more marked over the lower lobe.

Auscultation reveals a diminished respiratory murmur on the right side. Inspiration slightly harsher than normal, expiration somewhat prolonged.

Tuberculin Test.—Von Pirquet test (Koch's Old Tuberculin full strength), very slightly positive. Tuberculous infection present but not active.

Diagnosis.—Some thickening of the tissues in the right lung, probably as a result of pneumonia or of the attacks of bronchitis which patient has had in the past; probably peribronchial thickening. No active inflammatory condition present.

X-Ray Findings (See Fig. 99).—

Right Side.—Mild degree of peribronchial thickening extending from hilus of upper lobe into apex.

Middle lobe clear. A more marked infiltration, extending from hilus of lower lobe and reaching diaphragm, the surface of which shows an inflammatory thickening.

Left Side.—Clear, except small fibrous infiltration at hilus of upper lobe.

Case 2851.

Female, age 26 years, occupation—office work.

Clinical History.—One brother ill of tuberculosis at the present time. Patient was delicate until fifteen years of age, since which time she has been in fair health, but has had an unusual number of colds from which she always recovered slowly. She has had the ordinary diseases of childhood and several attacks of supposed malaria. For the past two or three years in the fall she has found it necessary to take a vacation in order to be able to continue her work. During the past year she has not felt as well as usual.

She has now, or has recently shown, the following symptoms:

I. Toxic Group.	II. Reflex Group.	III. Tuberculous Process *per se.*
Malaise	Slight hoarseness	Protracted colds
Lack of endurance	Tickling in larynx	
Loss of strength	Dry hacking cough	
Nervous instability		
Slight digestive disturbance		
Loss of a few pounds in weight		
Feeling of increased warmth, although temperature not increased, when taken		

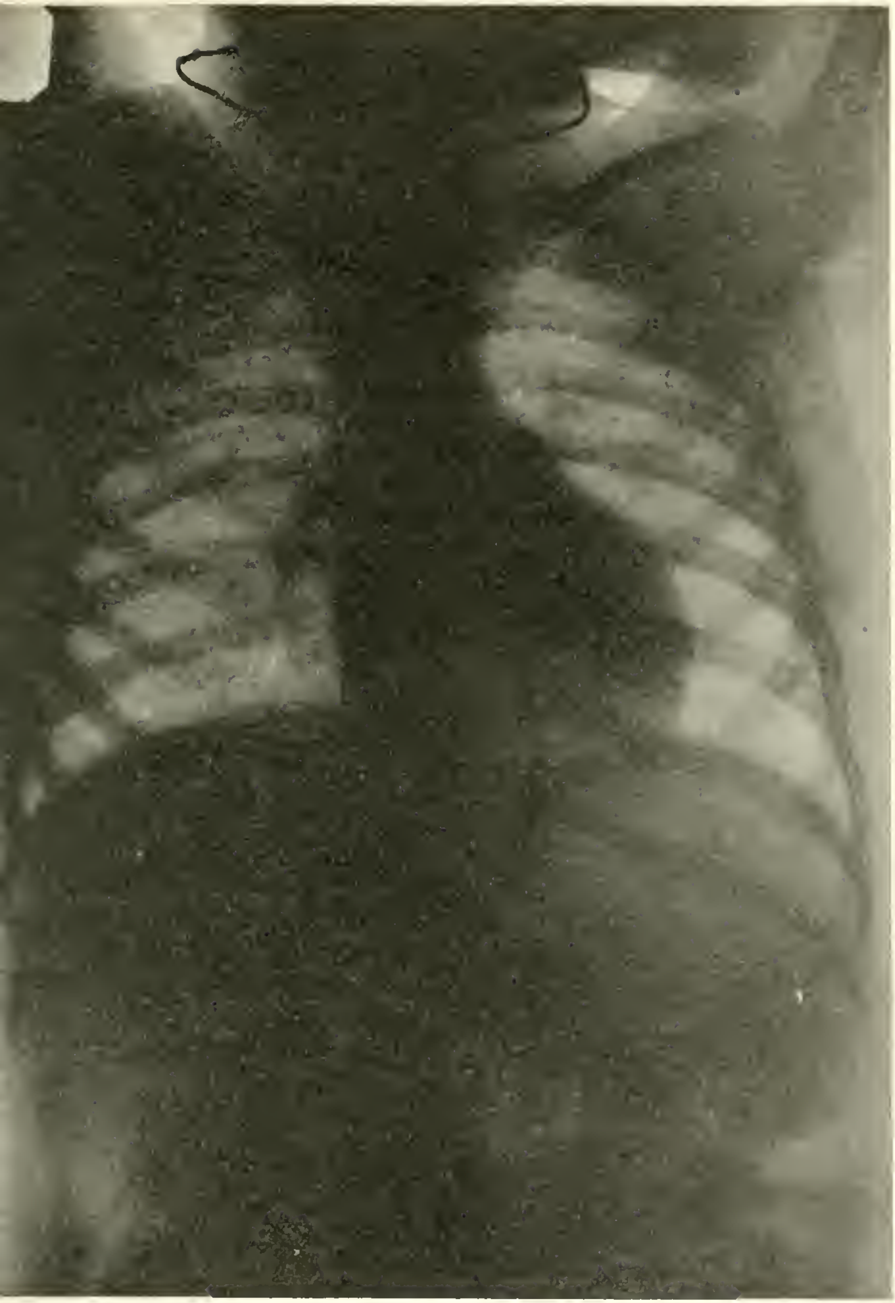

Fig. 99. Peribronchial thickening in a child six and a half years of age.

Physical Examination.—Inspection and palpation reveal the following: Chest considerably flattened over the apices. The soft tissues show a moderate degree of degeneration over both apices, most markedly on the right. This degeneration includes the skin, subcutaneous tissue, sternocleidomastoidei, scaleni and pectorales anteriorly and the trapezii and levator anguli scapulæ posteriorly.

Aside from the degeneration which is noted, the pectoralis, trapezius, and levator anguli scapulæ on the right show slightly increased tone (spasm). The sternocleidomastoideus, scaleni, pectoralis and trapezius on the left also show slight increased tone (spasm).

The right side of the chest seems to lag a little more than the left.

This degeneration of the muscles and subcutaneous tissue indicates a chronic inflammation, affecting both lungs, which is more marked on the right side than on the left. The increased tone (spasm) also indicates activity on both sides at the present time.

Deep palpation elicits increased density over both apices and over the fourth, fifth, and sixth thoracic vertebræ with the pulmonary tissue adjacent to them.

Percussion shows slight impairment of the note and increased resistance over both upper lobes, being about equal on the two sides. The resonance over the fourth, fifth, and sixth thoracic vertebræ is diminished and the resistance increased.

Auscultation over the right lung shows both inspiratory and expiratory notes harsher than normal above the third rib anteriorly and the spine of the scapula posteriorly. Expiration is slightly prolonged and here and there the inspiratory note is somewhat rougher than normal. Adventitious sounds which seemed to be extrapulmonary are present anteriorly near the apex. On the left side particularly the auscultatory note is slightly rougher than normal in the first and second interspaces near the sternum and above the spine posteriorly.

Tuberculin Test.—Von Pirquet test (Koch's Old Tuberculin full strength), positive in twenty-four hours, reaching a maximum of 1x1¼ cm. in diameter; a papule in the center, with slight redness surrounding it.

Diagnosis.—Hilus infection. Chronic infiltration of the upper lobe on the right side with slight activity. Chronic infiltration upper left, not as extensive as on the right, with some activity.

X-Ray Findings.—

Right Side.—Apex on inner aspect is infiltrated from hilus.

Lower half of upper and entire middle lobe show diffuse infiltration, with peribronchial thickening.

Lower lobe generally clear, with some mediastinal fibrosis.

Left Side.—Apex clear.

Lower half of upper lobe slightly involved.

Lower lobe nearly clear.

Case 2573.

Female, age 24 years, family history—positive.

Clinical History.—Patient first consulted me February 10, 1915, with the following history:

After birth of child four years previously, patient felt tired and unable to do her work without fatigue, and became markedly neurasthenic. Her appetite was fair, although not normal, lost $2\frac{1}{2}$ pounds in weight, noticed slight tickling in the larynx, with tendency to cough at times.

Examination at that time revealed a slight lesion in both lungs, more marked in the right, which seemed to be approaching quiescence.

She was instructed in hygienic living and asked to return in six months for reexamination, or sooner, if ill.

My second examination was December 13, 1915, ten months after previous examination. Three months prior to this examination the patient had suffered from a severe cold, followed by a bronchitis which persists to the present time. At the present she is suffering from the following symptoms:

I. TOXIC GROUP.	II. REFLEX GROUP.	III. TUBERCULOUS PROCESS *per se.*
General nervous depression	Hoarseness	
	Slight dry hacking cough	Tuberculous bronchitis
Lassitude	Aching in right shoulder	
Lack of endurance		
Nervous instability		
Temperature 99.8		

Physical Examination.—Inspection, palpation, and percussion reveal the following:

1. Lagging of right side.

2. Slight degeneration and slight spasm of the sternocleidomastoideus, scaleni, trapezius, and the levator anguli scapulæ, on the right side.

3. Degeneration of the subcutaneous tissue on the right to the 4th rib anteriorly and the middle of the scapula posteriorly.

4. Slight degeneration of the apical muscles and the subcutaneous tissue on the left side near the hilus, running up towards the apex.

5. Palpation of total density reveals increased resistance over both apices and the interscapular spaces.

6. Percussion shows slight impairment of the note and increased resistance on the right to the third rib anteriorly and to the spine of the scapula posteriorly and in the interscapular spaces near the hilus on both sides.

On auscultation the inspiratory note is slightly rougher and harsher than normal over the right apex to the third rib anteriorly and the middle of the scapula posteriorly. Breathing on the entire right side is weaker than on the left. The expiratory note slightly harsher than normal near the hilus on the left side.

Tuberculin Test.—Von Pirquet test (Koch's Old Tuberculin full strength), positive, reaching a maximum of 1 cm. in diameter in 24 hours.

Diagnosis.—Hilus infection with extension to adjacent tissues on both sides. Right, apical infection of lung and pleura, now active. Left side quiescent.

X-Ray Findings.—

Right Side.—Apex clear.

Middle lobe involved in an acute process. Two circumscribed areas of tissue, apparently necrotic at hilus of this lobe.

Lower lobe generally clear, with exception of small degree of fibrosis.

Left Side.—Apex clear.

Lower half of upper lobe has considerable peribronchial thickening.

Lower lobe clear.

Case 2789.

Male, coal dealer, age 44 years.

Family History.—Two brothers died of tuberculosis. Patient associated with one of them for a time during illness.

Clinical History.—Syphilis in 1908. Never showed any signs of activity after disappearance of first symptoms. For the past two years patient has been gradually losing strength. Has suffered from mild symptoms of toxemia,—malaise, lack of endurance, gradual loss of strength, nervous irritability, and has also had recurrent attacks of slight hoarseness.

Physical Examination.—Inspection shows that the left side of the chest lags. Slight spasm and marked degeneration of the sternocleidomastoideus, scaleni, pectoralis, trapezius, levator anguli scapulæ, and the soft tissues over them on the right side. On the left there is slightly increased tone (spasm) of the sternocleidomastoideus and scaleni and a slight degeneration and increased tone (spasm) of the trapezius, and levator anguli scapulæ. The fact of a slightly increased tone of the trapezius and levator anguli scapulæ on both sides is somewhat confusing, but history elicits the fact that the patient is using dumb-bells for fifteen to thirty minutes each day, so the increased tone may be due to hypertrophy from work instead of increased tone of reflex origin.

Deep palpation shows increased resistance over the upper portion of both lungs anteriorly and in the interscapular region running up toward both apices posteriorly and over the fourth, fifth and sixth thoracic vertebræ.

Percussion reveals slight impairment of the note and increased resistance to the palpating finger near the hilus, running up to the apex on both sides anteriorly and posteriorly; and also an impairment of the note and resistance to the percussing finger over the fourth, fifth and sixth thoracic vertebræ.

Auscultation shows the inspiratory note to be slightly rougher and slightly harsher than normal near the sternum and running up to the apex on the left side, the same posteriorly from the apex down to the middle of the scapula. The sounds are accompanied by a sensation of stickiness and some adventitious sounds in the nature of clicks that seem superficial. On the right side, above the third rib anteriorly and in the interscapular space and above the spine posteriorly the breath sounds are harsher than normal.

Tuberculin Test.—This was not given because patient had been receiving therapeutic injections of tuberculin for some time, which would vitiate the result.

Diagnosis.—Thickening of tissue about the hilus, with scattered foci through the upper part of both lungs; probably quiescent on right but more recent and not wholly quiescent on left. From history and examination impossible to tell exact nature. Might be tuberculous or syphilitic or both.

X-Ray Findings.—

Right Side.—Apex clear.

Entire middle lobe infiltrated with a mild degree of fibrosis.

Lower lobe clear, except at hilus.

Left Side.—Both lobes fairly clear with exception of hilus which shows a characteristic fibrosis.

Note.—There is a cystic degeneration of the ribs most marked in the right side and involving the posterior articulations.

X-Ray in the Diagnosis of Tuberculosis of the Intestine.—According to the law of the intestine as given on page 40 in Volume II, when there is a point of irritation in the intestinal canal there is a blocking of the intestine above and a hastening of its contents below this point. This has been observed by the use of the x-ray and utilized in the diagnosis of suspected tuberculosis of the intestine. When a test meal has been given, an area of irritation is suspected if the contents pass by a given point with unusual rapidity. This does not make a diagnosis of tuberculous enteritis, however, because the irritation might be due to other causes; nevertheless it is extremely suggestive when suspicious symptoms are present. This has been reported on by Archibald,[11] and Brown, Sampson and Heise.[12]

This method of examination offers hope of earlier and more certain diagnosis in suspected tuberculosis of the intestines, and should lead to a better and more successful therapy. It must not be taken, however, as calling for operative procedure in all cases. It rather emphasizes a fact that we should know from our postmortem and operative records that tuberculosis of the intestinal tract is frequently present even without producing symptoms and that it heals the same as does tuberculosis in other parts of the body.

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[11] "The Rôle of Surgery in the Treatment of Intestinal Tuberculosis," Transactions, National Association for the Study and Prevention of Tuberculosis, 1917, p. 117.

[12] "The Early Roentgen Diagnosis of Ulcerative Tuberculous Colitis," Lawrason Brown, Homer L. Sampson; Jour. Am. Med. Assn., 1919, lxxiii, p. 77.

CHAPTER XX.

LABORATORY METHODS.

By Joseph Elbert Pottenger, A.B., M.D.

The special pathology of tuberculosis, in favoring the development of various complications, together with the prolonged course of the disease, in providing greater opportunity for the entrance of non-tuberculous disease processes, places a great responsibility upon the clinician engaged in the management of tuberculous patients. Any aid which may be obtained from laboratory methods, enabling him to make earlier diagnoses and better prognoses under these conditions, will be gladly accepted. Laboratory methods have been much more useful in diagnosis than they have in prognosis, in fact their relation to the formation of the latter judgment, is almost entirely confirmatory. In an indirect manner they offer assistance the value of which cannot be overestimated. They play an important part in keeping the confidence of the patient and giving him assurance, which is so necessary in the management of his case.

The choice of laboratory methods naturally depends upon the stage of the disease, particularly in regard to the examination of sputum. However, there are certain routine tests which may be made at regular intervals on urine, blood, and sputum, if present, regardless of the stage of the disease which fully repay for the time consumed in making them. Occasionally, traces of albumin and casts, and rarely a glycosuria will be found, in patients where no history of such, either past or present, may be elicited.

As 78 per cent of all patients who have entered the sanatorium in the last ten years were in the third stage of the disease, expectoration was usually considerable and bacilli plentiful, affording splendid opportunity to study the most characteristic material associated with the disease. If, then, the methods of analysis described under sputum seem to take too much space, as compared with the methods described under urine, blood, and feces, it is due to the class of patients that come to us.

SPUTUM.

The usual method of measuring the twenty-four hour quantity of sputum in hospital and sanatorium practice consists only in a rough guess as to the amount of pus, bronchial and pharyngeal mucus and saliva combined. No attempt is made to determine the proportion of these elements, but descriptive terms, such as mucoid, purulent, and mucopurulent are employed in describing the solid elements.

It occurred to us that a more exact quantitative determination of some of these elements might give valuable information and aid in interpreting the many complications likely to arise in the course of the disease. The total nitrogen determination was tried, but was found too time consuming for routine procedure, besides it gave no information as to the relation of the individual elements. As is well known, but too often forgotten, the total amount of expectoration varies greatly from day to day, due to many objective and subjective influences; so that little is learned from it usually, because of our inability to determine these influences. On the other hand, the cellular elements are fairly definite in amount from day to day and correspond quite accurately to the active process in the lung. I developed a technic for the measurement of the volume of the formed elements in 1911 and since then have made nearly five thousand determinations. After bacilli have once been found there is no other single test which gives more information with as little labor.

Our routine sputum examination is made every four or five weeks, just previous to the physical examination, and consists in the accurate measurement of the twenty-four hour quantity, or a three day quantity in case the bacilli are known or are supposed to be rare or absent, the cytological examination, the determination of the sediment volume, rough quantitative albumin test, the search for bacilli, and their morphological classification, if present.

Collection of Sputum.—The 24 hour specimen of sputum is collected, using a 150 c.c. glass jar with metal screw cap, which can be sterilized at 170 to 180° C. The patient is instructed to start at 8:00 o'clock at night and to save all material, whether or not he thinks it comes from the lungs, throat, or nasopharynx, until 8:00 o'clock the next night. A crystal of menthol is added in warm weather to deodorize. Special caution is given to avoid getting food particles in the specimen.

As these instructions are somewhat at variance with those usually given by physicians, I wish to indicate the advantage which they secure over those commonly given,—to save "only what comes from the lungs." Our experience leads us to believe that patients generally cannot be trusted in this matter. We constantly meet patients who state positively that they do not raise anything from the lung, but only from the throat and nasopharynx, while the specimens collected as above described, show typical cavity sputum, with plenty of bacilli. In quite a few instances we have found bacilli in sputa where they had not been found, although they had been searched for only a few days before by other laboratories. This is attributed to faulty instructions to the patients in collecting, rather than to any special superiority of technic.

The instructions to save "only what comes from the lungs" are based upon the fear that pus, epithelium, or mucus from other parts of the respiratory tract may so dilute the bacilli that they may not be found if present in small number. This danger is much overrated. Postnasal and pharyngeal mucus in simple catarrhal conditions is destroyed by all modern methods of examination, such as the antiformin or the fermentation methods, with their various modifications, and the formed elements associated with such mucus is inconsequential. A small amount of saliva mixed with the sputum, I consider advantageous, as it aids the fermentation process which is employed. Only rarely do I meet a case where it seems advisable to modify these instructions, on account of the presence of a posterior suppurative rhinitis. In such case, the patient is taught to keep such material from the specimen.

In order to obtain exact results it is absolutely necessary to avoid the two hours following the awakening of the patient, as the time at which to start and stop the collection of the specimen. The time for expectorating depends largely upon the position and activity of the patient. Most patients after a night's rest feel the irritation of the accumulated sputum as soon as they awaken and change their position in bed or arise on their feet. Within the next hour or two they will have raised all that has accumulated, and may raise little if any from the lungs during the day. Many patients raise again at night on changing position on retiring. Excluding those cases with massive involvement, where the inflammatory products accumulate rapidly and are raised constantly, it is probably correct to say that on an average, three-fourths of all the solids of the day's expectoration is raised within the two hours following the awakening of the patient. Now, as the awakening time of the patient varies from day to day, should the specimen be started during the period of active expectoration, there is great likelihood that the supposed twenty-four hour specimen would represent more or less than the amount secreted in twenty-four hours, depending upon whether the patient arose earlier or later on the second morning than he did on the first. For this reason, the plan of saving from night to night is preferable.

Cytological Examination.—A study of the formed elements had best be made from fresh material, though in comparing preparations from the twenty-four hour specimen and from fresh morning expectoration, I have been unable to see any difference unless fermentation has been marked in the former specimen. Neutrophiles from tubercular cavities usually stain poorly, whether in fresh or older specimens. The protoplasm stains indistinctly, the nucleus is shrunken, and the cells often simulate lymphocytes. Their appearance, in my experience, is in marked contrast to neutrophiles found in the sputa of certain acute respiratory infections. Here the contour of the cells, their protoplasm and nuclei are all distinct and well differentiated. There is great inequality in the distribution of the various cells in sputum. Yellow or green sputa always contains a predominance of neutrophiles, or less commonly of eosinophiles as in asthma. Gray sputa contain epithelium, both pavement and alveolar, and lymphocytes in predominance. One should take several distinct particles on the slide and examine them separately, since a particle originating from an old healing cavity or from the region of a tubercle before softening has taken place is likely to be composed chiefly of lymphocytes, while, after cavitation has taken place, neutrophiles predominate. One can hardly make a satisfactory differential count of the cells because of the uneven distribution. A rough estimate is all that can be expected. Lymphocytes are likely to be confused with certain small forms of epithelia, and certain degenerated mononuclear neutrophiles, and only continual observation will give confidence in their differentiation. I have employed Hasting's and Giemsa's stains, preferably the latter. After drying the smear, flood with the stain made in the proportion of one drop to 1 c.c. of distilled water. A longer time is required to stain than is the case with blood films,—20 minutes to one hour giving best results.

Fermentation and Determination of the Sediment Volume.—Having measured the total amount of sputum for 24 hours by direct comparison with a graduated bottle of the same size, the specimen is roughly homogenized by shaking, either in a mechanical shaker for two to five minutes, or by hand. Prolonged shaking is to be avoided, as it interferes with sedimentation. A small portion of the sputum is then placed in a graduated cylinder or any cylindrical tube with flat bottom. Homeopathic vials 8 to 10 cm. long and of 10 c.c. capacity are well suited to this purpose. The cylinder or vial is then tightly corked and placed at 37°. In purulent sputa, the mucus is completely dissolved within 12 to 24 hours; but 48 hours is required in the case of some mucoid sputa. The cellular matter then settles more or less compactly to the bottom. This settling continues slowly until about the fifth day, after which scarcely any change is observed, even after two or three weeks. The proportion of sediment to the whole amount is then determined from the graduate, or from the vials, by means of a rule graduated in mm. The total sediment for the 24 hours is then found by proportion. This reading is called the gravity sediment volume.

Formerly this reading was accepted as the standard, but owing to very large errors met with in about 25 per cent of the readings made, I recommend the use of the centrifuge. A rather high power centrifuge is required. At present the standard condition for operating the centrifuge is 30 minutes at 2800 revolutions per minute, with a tube length of 7.5 inches. One hundred and three findings obtained by the 5-day gravity method were compared with the readings after centrifuging the same tubes. It was found that on an average the reading obtained by the gravity method was reduced to 51 per cent by the centrifuge so that the approximate factor for converting the centrifuge reading into the gravity equivalent is 2. Comparisons between centrifuge readings taken at 30 minutes and at 60 minutes showed only a 10 per cent reduction in volume.

In order to overcome the objection to such a method of analysis because of the time required, I have employed a shorter and more practical method with some degree of success. The specimen is placed in a water bath at 52° for from 6 to 24 hours, and then centrifuged, giving readings very close to those obtained on duplicates at the end of 5 days fermentation.

The determination of the sediment volume is recommended with a knowledge of the well known errors in centrifuge methods, when quantitative results are attempted. The error is under 10 per cent in one-half of the specimens examined; between 10 and 20 per cent in one-fourth; and over 20 per cent in the remaining one-fourth. On the other hand, there is a great difference in the amount of the sediment volume as met with in different patients, and in the same patients at different times, due to different degrees of activity of the disease; so that a considerable error may be allowed. My readings range from less than 1 c.c. to 105 c.c. The latter was in a case of tuberculosis complicated with pulmonary abscess. The sediment volume readings may be placed in several fairly definite groups, representing the degree of activity and extent of involvement in the lung. Less than 5 c.c. is found in early cases without bacilli, or if bacilli be present, without general symptoms. From 5 c.c. to 15 c.c. represents a moderate degree of activity and involvement. Fifteen c.c. to 30 c.c. indicates severe activity, massive involvement, or both. Above 30 c.c. is rarely met in uncomplicated tuberculosis, lung abscess being the condition met most commonly.

Albumin Reaction.—The original technic of Roger for the detection of albumin in sputum does not readily give accurate quantitative results; for one cannot conveniently eliminate bubbles of air which are more or less mixed with the sputum. In order to properly correlate our data, fairly accurate relative quantitative determinations were considered essential.

The first problem to be solved is the removal of the mucus. This is accomplished by most workers in the following manner: The sputum is diluted with 2, 3, or 4 parts of .6 per cent salt solution and thoroughly shaken. A weak solution of acetic acid (2 to 5 per cent) is added drop by drop until the filtrate is clear, and does not give a cloud with the acetic acid. The filtrate must be neutralized carefully with sodium hydrate before testing. At this point, the worker is left to choose such method as he may desire for the albumin test.

Albumin behaves in a very uncertain manner in the presence of acetic acid as a demucinizer unless one is very careful to neutralize. Even then there appears to be some loss of albumin—the loss increasing as the time and amount of agitation increases before filtering. In order to avoid this difficulty I have relied upon the fermentation process as the most convenient demucinizer. Most purulent sputa will, after 6 hours standing at 37°, filter slowly but clearly through good filter paper without previous diluting. I have made comparisons of the albumin found in filtrates by this method, with the albumin from filtrates in which acetic acid was used to destroy the mucus, and have found that the reaction is invariably more distinct by the fermentation method. There was an estimated increase of 10 to 25 per cent by the heat-salt-acetic acid and nitric acid-contact tests. On the other hand, if fermentation proceeds too far the albumin is split and there is a marked loss at the end of 24 hours. This loss continues for several days. The dilutions were made accurately by weight whenever it was necessary to work with fresh specimens containing air bubbles.

The following method may be used, giving relative and fairly accurate quantitative results: Allow sputum to ferment to the point where the sediment begins to settle. Filter the supernatant liquid. Draw up 1 c.c. of the filtrate in a pipette and dilute to 10 c.c. with 1 per cent salt solution. To 6 c.c. of the dilution add saturated salt solution to 7.5 c.c. After mixing, the solution is poured into a half-inch test tube. The upper portion is then heated to boiling, three drops of glacial acetic acid are added, and heat is again applied. The strong acid is used because it favors the development of characteristic columns descending into the unheated portion. A weak acid does not do this. The appearance of these columns at the end of 20 seconds is used as the indicator. If the reaction is heavy at $\frac{1}{10}$ dilution, the columns appear at once; if lighter, they appear at, say 5 or 10 seconds; if still lighter, at 20 or 30 seconds, etc. One can soon judge from the intensity of the reaction in the $\frac{1}{10}$ dilution just how much further it should be diluted in order to give the end reaction at the end of 20 seconds. One per cent albumin gives this reaction at the end of 20 seconds in a dilution of approximately $\frac{1}{100}$. The albumin content of saliva never gives the reaction beyond $\frac{1}{10}$ dilution, usually at only $\frac{1}{5}$ dilution. Thus a positive test at any dilution above $\frac{1}{10}$ may be considered pathological. The relative 24-hour albumin content is determined by multiplying the dilution recorded by the number of c.c. of sputum in the 24-hour quantity.

Studies on the Distribution of Tubercle Bacilli in Sputum and Other Conditions of Importance for Their Demonstration.—Although many advances within recent years have been made in the early recognition of tuberculosis before bacilli may be found in the sputum, yet there is a considerable percentage of borderline cases in which doubt as to diagnosis prevails, even after careful physical examination, tuberculin tests and x-ray plates have been made, and an accurate case history has been taken. Consequently, it is imperative to develop and employ such technics as offer us the greatest chance of finding tubercle bacilli in the sputum.

Of the many known technics for this purpose, little is known of their comparative value, and until some method is developed for their accurate standardization, each method will continue to have its supporters. Most authors in proposing their technics, have presented the claim to superiority by comparing the percentage of positive results obtained in a number of specimens, with the percentage obtained in fresh smears from the same specimens. The technic which is likely to attract most attention, then, is the one, the author of which reports the highest percentage of positive findings as compared with findings in direct smears. Such evidence is not valid in establishing the relative efficiency of technics in general, unless all workers search for the same length of time. The time spent in searching specimens is rarely mentioned by writers, although it is the easiest factor standardized. It is not probable that workers vary greatly in the speed with which they examine a stained preparation, and as the chance of finding bacilli increases with the time spent in searching, regardless of the technic, it seems strange that this factor should have been almost completely overlooked.

There are a variety of other factors and conditions essential to a good technic, some of which admit of approximate standardization with little or no extra demand upon the worker, except the time required to make a few notes. Uniformity of thickness of preparation, presence or absence of dust particles, scratches on slides and artifacts, nature of the background provided by the counterstain and the penetration of stain, all contribute to the success or failure of a technic. More important perhaps than all other conditions combined, is the uniform distribution of bacilli in a preparation to be examined. The varying results reported by different workers employing the same technic are undoubtedly due to failure to appreciate the importance of these little details; and there has been too much tendency to credit the "personal equation" with the resultant successes or failures.

The following pages set forth the result of an inquiry into the importance of some of these conditions. This work was done entirely by daylight, and the time of search was 15 minutes for each preparation. The mechanical stage was employed and the distance traversed was recorded. A total of 120 specimens were examined and classified according to whether they contained less than 5, 5 to 20, 20 to 50, 50 to 100, and 100 or more dust particles in each field. It was found that the average distance covered in 31 clean preparations was 13 cm., and in 4 preparations containing 100 or more particles, the average distance was 8 cm. This is a very appreciable loss of efficiency. It is also a condition very trying on the eyes.

Maximum penetration of stain in the bacilli is not always easy to secure. There are a number of influences working against it, such as the presence of food particles, especially fat, in the sputum, and the effects of heat and chemicals used in the process of homogenizing and staining; so that however careful one may be in securing standard conditions for the staining of preparations, such as uniform degree of heat and definite time for exposure to stain and decolorizing reagents, there are still marked differences observed in the staining of the same strain of bacilli when compared after various methods of treatment. Bacilli exposed to as low as 1 per cent NaOH for a short time show lessened penetration of stain.

For practical purposes, in comparing the staining of the bacilli under different conditions, I classify them into three groups, deeply stained, well stained, and pale. The lines of separation are, of course, somewhat indefinite, but with Spengler's staining method, to be described later, and which I have used for eight years, I have obtained more uniform results than I had obtained before that time by the older methods. In deeply stained bacilli met in the thinnest preparations the beads, even when present, are barely visible or are not seen at all. Passing to thicker parts of the preparation the bacilli become narrower, less deeply stained, and the beads stand out prominently. These are called well stained bacilli. Still thicker parts will show very pale pink bacilli. These are called pale. I never make a diagnosis from the presence of pale bacilli alone, but call them questionable.

The following table illustrates the comparative chances of making a diagnosis in a specimen if the time of search were 1 minute, 2 minutes, 5 minutes, 10 minutes, and 15 minutes respectively. The specimen was selected because of the rarity of bacilli and was searched for two and one-half hours. Each bacillus and bunch was recorded with the minute during which it was found. The preparation was made after fermentation and shaking for 10 minutes.

In order to simplify the mathematics let us assume that at the end of the 10th period or the 150th minute, the same microscopic field appeared as with which we started. We will then have 150 possible consecutive periods, of 15 minutes each, to consider. The first column of findings represents the first period. The 2nd minute up to and including the 16th minute represents the second period. The 150th minute up to and including the 14th minute represents the last possible period.

TABLE I.

Detail Findings in Sputum with Rare Bacilli Searched for Two and One-Half Hours—Divided into Ten Periods of Fifteen Minutes Each

Min.	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
1...	1	0	0	0	0	0	0	1	0	0
2....	1	2	0	0	0	0	0	0	0	0
3...	0	1	0	0	0	0	0	0	0	0
4...	0	0	0	0	0	0	0	0	2	0
5..	0	1	0	0	0	0	0	0	0	0
6...	0	0	0	0	0	0	0	0	0	0
7.	0	0	0	1	1	0	0	0	0	0
8..	0	0	1	0	0	0	0	0	0	1
9.	0	0	0	0	0	0	0	0	1	0
10	1	8	2	0	2	0	0	0	0	0
11..	0	0	0	0	0	0	0	0	1	0
12..	0	0	1-1	0	0	1	0	0	0	0
13..	0	0	0	3	3	0	0	1	0	1
14.	0	1	0	0	1	0	1	0	0	0
15.	0	0	0	0	0	1	1	1	0	0

Inspection of Table I shows that at least one bacillus or bunch of bacilli was found during each possible consecutive 15 minute period. Considering possible consecutive 10 minute periods, a diagnosis was made in 139 and no diagnosis in 11. With 5 minute periods bacilli were found in 100 and were absent in 50. The 2 and 1 minute periods were positive 53 and 29 times respectively.

TABLE II.

Tabulation of Positive and Negative Periods of 1 Minute, 2 Minutes 5 Minutes, 10 Minutes and 15 Minutes Each, from Data Supplied by Preceding Table

	1 minute	2 minutes	5 minutes	10 minutes	15 minutes
Positive periods................	29	53	100	139	150
Negative periods..............	121	97	50	11	0
Per cent chances of finding....	19.3	35.3	66.7	92.6	100

As there are many sputa in my experience which contain approximately the same number of bacilli as the one above, the time of search is evidently of much importance. This is further shown in the following summary of all records of rare bacilli in which there were one or more minute periods during which no bacilli were found. Table III presents 147 such records from 54 different patients. One hundred and nineteen of these records were made from preparations after fermentation and shaking in a mechanical shaker. Of the remaining 28 some were made from sediment precipitated by alcohol after shaking fermented specimens with antiformin of 15 to 20 per cent strength, a few after shaking fresh sputum in the shaker, and a few after fermentation without shaking.

TABLE III.

SHOWING CHANCES OF FINDING TUBERCLE BACILLI WITHIN VARIOUS PERIODS OF TIME, WHEN BACILLI ARE RARE

Method of Preparation	Number of Sputa examined	1 minute period	2 minute period	5 minute period	10 minute period	15 minute period
Fermented..........	14	8	8	13	14	14
Fermented; Shaken 10-20 min.........	119	37	58	88	110	119
Fermented; Shaken with antiformin....	3	2	2	3	3	3
Shaken fresh 10-20 min..............	9	4	5	7	9	9
Miscellaneous.......	2	....	....	1	2	2
Total...........	147	51	73	112	138	147

In choosing a time standard, in searching for bacilli for diagnostic purposes, clinicians will vary considerably according to the importance which they place upon the presence of rare bacilli. In comparing the different time standards, we must consider the average amount of time consumed for each additional diagnosis made. That 15 minutes is the maximum time standard and that little is gained in searching beyond that point at enormous expenditure of time, is shown in the results obtained in another series of 120 specimens which were found negative after 15 minutes' search. Thirty of the preparations were examined for a second 15 minute period. New preparations were made from 13 of the specimens and they were searched for 15 minutes. A third series of 77 preparations was made after treating the sediment with antiformin, and examining for 15 minutes. Each series gave one positive result, or 3 in all.

It is evidently impracticable to prolong the search to 30 minutes, since in order to obtain 3 diagnoses the amount of time consumed was 120x15 minutes, or 30 hours of hard labor; that is 10 hours for each diagnosis. Rather than search beyond 15 minutes, these detailed studies, particularly as shown in Tables IX, X, and XI, indicate that time would be saved by calling for another specimen. If we compare the 10 and 15 minute time standards, as shown in Table III, we find that nine additional diagnoses were made in favor of the latter, at an expenditure of 147x5 minutes, or 735 minutes; that is, 82 minutes for each diagnosis. In the same way, the time required to make each additional diagnosis obtained in 10 minutes, as compared with 5 minutes, is 28 minutes; in 5 minutes as compared with 2 minutes, is 13 minutes; and in 2 minutes, as compared with 1 minute, is 7 minutes.

The great lack of uniformity in distribution of bacilli in sputum is well known, but apparently quantitative studies have not been reported. In order to obtain accurate information on this subject, four years ago I arbitrarily established 15 minutes as my standard for searching a preparation before calling it negative, and have continued this practice until the present time. The time was determined accurately by watch. In case the bacilli were rare, averaging one or less per minute, 15 minutes was usually employed, and each bacillus or bunch, the latter expressed by the approximate number of bacilli contained therein, was recorded with the minute during which it was found. A note was made also as to whether the bacilli occurred in the same field. Detailed records of this sort have been made of which Table IV (A and B) are illustrations. Further studies were made on strongly positive specimens, to ascertain the degree of bunching of bacilli. Five hundred free bacilli, with the accompanying bunches, were usually counted, requiring from 5 to 10 minutes. Table V illustrates the form in which the detail was recorded.

Counting the free bacilli by hundreds, we obtain an idea of the accuracy of the differential count. In a total of 54 consecutive differentials, 37 showed a probable error (computed by least squares) within ± 10% ; the remaining 17 exceeded ± 10%.

TABLE IV.

Form of Record Where Bacilli Are Rare

Minute	Sputa A Bacilli	Sputa B Bacilli
1	1	0
2	0	0
3.	0	7
4	0	20
5	1	0
6.	0	1
7.	1	3
8.		0
9.	0	0
10.	0	0
11.	1	2
12	0	0
13.	5	0
14	1	0
15.	$\overline{1-1}$	0
Total Bacilli	13	33

The dash over the findings in 15 minute sputum A indicates that they were found in the same field but did not touch each other

TABLE V.

Form of Record for Recording the Distribution of Bacilli

Free Bacilli	Bunches Of						Bacilli In Bunches
	2	3	4	5	6	Etc.	
100	15	2					36
100	18	3	1				49
100	13	5	0	2	1		57
100	18						36
100	14	2					34
Total 500	78	12	1	2	1		212

Total Bunches.....	94
Total Bac. in Bunches..	212
Total Bacilli.....	712

The summary of these differential counts are presented in Tables VI and VII. The former presents 174 records from 73 patients, in which bacilli were present one or more per field. The latter presents 115 records from 47 patients in which bacilli were less than one per field. Most of the sputa were shaken fresh for 10 minutes in a mechanical shaker. Some others were shaken for 10 minutes after fermentation, while a few others were examined after shaking by hand. Tables VI and VII show no appreciable difference in the averages after the various methods of

TABLE VI.

SUMMARY OF DISTRIBUTION OF BACILLI AFTER DIFFERENT METHODS OF PREPARATION WHERE BACILLI WERE MORE THAN ONE PER FIELD

Method of Preparation	Specimens examined	No. bacilli free	No. bacilli in bunches	No. bunches	Total bacilli	Total free bacilli + bunches
Fermented, Rough shake...	19	12250	19052	6407	31302	18657
Fermented, Shaken 10 min.	9	5800	9624	2177	15424	7977
Shaken fresh 5-10 minutes...	139	69020	50698	18430	118718	87450
Shaken fresh by hand	7	4625	5034	1585	9659	6210
Total.....	174	91695	84408	28599	175103	120294
Ratio .. .						1 455

TABLE VII.

SUMMARY OF DISTRIBUTION OF BACILLI AFTER DIFFERENT METHODS OF PREPARATION WHERE BACILLI WERE LESS THAN ONE PER FIELD

Method of Preparation	Specimens examined	No. bacilli free	No. bacilli in bunches	No. bunches	Total bacilli	Total free bacilli + bunches
Fermented, Rough shake . .	20	530	1328	311	1852	841
Fermented, Shaken 10 min..	68	1329	2143	676	3472	2005
Shaken fresh 5-10 minutes...	25	1183	1359	413	2542	1596
Antiformin.....	2	20	12	6	32	26
Total........	115	3062	4842	1406	7898	4468
Ratio........						1 768

treatment, except that a few of the fermented specimens in both tables were selected because of the presence of bunchnig to an extreme degree. Consequently, the number in bunches is much greater than is found in the specimens prepared by other methods.

From Tables VI and VII it is noticed that the ratio between the total bacilli and the combined total of free bacilli and individual bunches is respectively 1.455 and 1.768, which indicates a corresponding loss in efficiency in finding bacilli. If complete resolution of these bunches into their component cells could be attained, with subsequent thorough homogenization, the chances of finding the first bacillus within a certain period of time would increase about 45 per cent and 77 per cent respectively. This is important for the specimens summarized in Table VII, because most of them present some difficulty in diagnosis. Referring to Table IV, Sputum B, which is an illustration of one of the specimens, it is seen that with complete resolution of bunches there would be 33 chances of making a diagnosis in 15 minutes, which with perfect distribution should give a fraction over 2 bacilli during the first minute. As a matter of fact there are only 5 chances of making a diagnosis—a loss of 85 per cent efficiency, due to bunching.

TABLE VIII.

Showing Solvent Effect of Various Reagents Upon the Interbacillary Wax

	Free Bacilli	Bunches	Bacilli in Bunches
Shaken 3 minutes	1000	310	2084
Shaken 20 minutes	1000	373	1740
Ether 2%	1000	269	850
Xylol 10%	1000	35	76
Ligroin 10%	1000	65	146
Chloroform 10%	1000	38	78
Carbon bisulphide 10%	1000	42	101
Antiformin 20%	1000	164	836

Three years ago I conducted some preliminary experiments to determine the effect of various solvents upon the interbacillary wax, with the view of setting the individual bacilli free. A fermented specimen was chosen which showed bunching of bacilli to an important degree. The sputum was shaken 3 minutes in the shaker to obtain rough homogenization. To various portions were added reagents, as in the preceding table. All portions were then shaken for 20 minutes. Differential counts were then made of the free bacilli and bunches.

The table shows clearly that xylol and chloroform secure almost perfect resolution, with carbon bisulphide and ligroin next in importance. Ether and antiformin are far behind in efficiency. Shaking for 20 minutes without reagent gives only slight resolution.

Further studies were made to ascertain the chances of finding bacilli on any particular day in a series of successive daily examinations of 24 hour specimens from patients in whom bacilli had previously been found to be rare. Three separate series were studied from as many patients. Fourteen examinations were made on the first, with one negative result. Seventeen were made on the second, with three negative results. The positive findings in these two patients were such as would give no probable difficulty in making a diagnosis by methods which do not secure so uniform a distribution. The third, however, because of his previous history together with the record I had made, seemed to present a difficult problem in diagnosis. His laboratory record before coming to the institution is as follows: Within three weeks before entering the sanatorium he had saved morning specimens ten different times, without a diagnosis having been made. The technic used has not been ascertained. Within three days before entering a few bacilli were found by another laboratory at the end of two hours' search. My record of this case, prior to the detailed experiment, is as follows:

TABLE IX.

Oct. 26, 1913	Nov. 22, 1913	Dec. 28, 1913	Jan. 30, 1914	Apr. 2, 1914	May 10, 1914
3 day spec.	3 day spec.	3 day spec.	3 day spec.	3 day spec.	3 day spec.
1 bac. 1st min. 2 bac. 15th min.	Negative	Negative	2 bacilli 15th min.	Negative	Negative

Because of this history of rare bacilli alternating with negative results, this patient was chosen for the experiment indicated above. The record of daily sputum findings for the 27 successive days was kept as follows:

TABLE X.

RECORD OF DAILY FINDINGS IN SPUTUM OF PATIENT, WITH RARE BACILLI

	June, 1914													
	18	19	20	21	22	23	24	25	26	27	28	29	30	
Bacilli found within 15 minute search	1	2	1	0	1 2	1 4 1	0	7 1	0	0	0	0	0	
	July, 1914													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Bacilli found within 15 minute search	0	1?	1 2 2 2	0	0	0	0	1	0	1-3 2-1 1 1 1	0	1 1-1-1 1 1	0	0

Figures separated by hyphen mean that the bacilli were found within the same minute period, but not in the same field.

The above table illustrates two important points; first, the difficulty in finding bacilli in this case; second, the superiority of the 3 day specimen over the 1 day specimen as a time saver. It has been my uniform practice to have patients save everything for a period of three days in cases where bacilli have never been found, or, if found were rare.

Inspection of the above table enables us to tabulate the fol-

TABLE XI.

SHOWING RELATIVE PERCENTAGE EFFICIENCY IN THE DEMONSTRATION OF TUBERCLE BACILLI OF TWENTY-FOUR HOUR AND THREE DAY SPECIMENS

	Total Specimens	Negative	Positive	Chances of finding bacilli on any given examination
24 hour.. ..	27	17	10	37.0%
Possible 3 day	25	12	13	52.0%
Distinct 3 day.	9	3	6	66.7%

lowing, showing the relative efficiency of a 24 hour, and 3 day specimen. We may represent this relative efficiency by considering (a) all possible consecutive 3 day specimens, and (b) each 3 day specimen as distinct. The first method presents the series 18, 19 and 20; 19, 20 and 21, etc.; and the latter method the distinct series, 18, 19 and 20; 21, 22, 23, etc.

In the other two patients mentioned above, the 3 day specimen would have given 100 per cent findings in both, whether considered as possible, or distinct 3 day specimens.

Technics for the Preparation of Sputum.—The choice of a technic for the preparation of sputum depends somewhat upon the character of the specimen to be examined, upon whether other tests are to be made, and upon the urgency of diagnosis. In sanatoria time is usually of less importance, so we may employ the slower but more certain methods. All workers are agreed that the fresh smear is inadequate if the result is negative. But as to the choice of the next method to be adopted no uniformity of opinion exists.

It seems to me that the most sensitive technic must secure the following ends, each capable of demonstration, and each attained without injury to the bacilli: (1) resolution of all bunches of bacilli into free independent cells; (2) distribution of the bacilli uniformly through the cellular matter; (3) maximum concentration of bacilli. The importance of distribution and concentration have long been emphasized by workers; but I am not aware that any one has called attention to the importance of the resolution of bunches.

Ellermann and Erlandsen, for the first time, demonstrated by exact quantitative methods the degree to which it is possible to concentrate bacilli by the various technics in use at that time. Starting with the theory that in the search for bacilli concentration is the great end to be attained, they based their conclusions as to the comparative value of technics upon the relative number of bacilli found in unit volume of sediment obtained by centrifuging for one hour at uniform speed. Their work is the first serious attempt to standardize technics. They established beyond all doubt the importance of the viscosity and specific gravity of the material to be examined in preventing concentration by means of the centrifuge. Their method of analysis secures the following desired ends: Lowering of viscosity to almost that of water; lowering of specific gravity to 1006 to 1008; almost complete solution of pus and other formed elements. The method gave, in their hands, from three to fifteen times as many bacilli as were found in the smears of the same thickness made by other methods. They homogenized the material used for comparison by prolonged grinding in a mortar.

My experience, as suggested by the studies above, has convinced me that of all single ends to be desired homogenization is by far the most important—more important even than concentration. Ellermann and Erlandsen avoided the necessity of discussing the importance of homogenization, by assuming it.

It must not be forgotten that bacilli in sputum are extremely irregular in distribution; one unit portion containing practically no bacilli, another containing thousands, still another millions; and yet these different portions may be quite similar in appearance. Every laboratory worker in examining fresh smears has searched many fields in some specimens without result, and then suddenly found a number of bacilli lying near each other. Concentration is not the important point in this instance. In fact, the bacilli are too concentrated. If they were uniformly scattered, a much less number of fields would need be searched in order to find a bacillus. Fortunately, all the best modern methods secure homogenization to an important degree before the centrifuge is used for concentrating; but the centrifuge gets credit for too large a share of the increased efficiency of such technics. By far the greater part of such efficiency is due to the homogenization attained by brisk shaking and the solvent action of chemicals.

The Direct Smear.—In my own practice the fresh smear is rarely employed, as bacilli have been found in all but a small number of patients, before entering the sanatorium. When called upon to examine fresh material, I select with a glass hook a small portion from fifteen different parts of the specimen, add one drop of one per cent NaOH, and homogenize by drawing one glass slide over another 50 times. A drop of water is then added and thoroughly mixed with the material; the slide is tilted and dried on an oven at 60°. Slides made in this manner give excellent distribution of bacilli, and materially reduce the necessity of using the more complicated technics. Of late one drop of xylol has been added with the sodium hydrate. The resolution of bunches determined by differential counts is almost as thoroughly attained as with xylol and the mechanical shaker.

The old fermentation method of Phillip has served as the basis for my routine methods. He observed that if sputum, negative on direct smear, be placed at 37° for 24 hours, fermentation takes place, the mucus is destroyed and bacilli are often found in the sediment. It was formerly considered that the bacilli multiplied in the incubator, and Nuttall came to this opinion, from results obtained in making exact counts. He made daily counts on 24 hour specimens; those obtained on the first, third, fifth, etc. days, were counted fresh, and those obtained on the second, fourth, sixth, etc. days, were counted after fermentation. This indirect method is hardly convincing, unless a much larger number of counts be made than he reported. His reason for not making the counts on the same specimen when fresh and after fermentation, was that the violent shaking necessary to secure homogenization of the fresh specimen, might so injure the bacilli as to destroy their reproductive power. His counts on the fermented specimens average a little higher than those on the fresh specimens. I have made counts on three different specimens when fresh after thorough homogenization, and again on the same specimens after fermentation; the first was 5 per cent greater, the second 4 per cent less, and the third 7 per cent greater, after fermentation than when fresh. These results fall within the range of the probable error of the counting method, so that there is no evidence of growth. On the other hand there may have been slight growth had the experiment been conducted as Nuttall suggested.

The Phillip method has not come into general use, perhaps because of the necessary delay before making the preparation; although it is considerably superior to the fresh smear method. I adopted it years ago because it often gave positive results when the fresh smear had been negative. Its superiority over the direct smear is due probably almost entirely to better distribution of bacilli.

Impressed with the importance of thorough distribution of bacilli, following the publication of Rickard's article advocating the use of the mechanical shaker, I have employed this method almost exclusively on all doubtful specimens and on those specimens from patients who previously gave few bacilli. On account of the lowered viscosity due to fermentation, better distribution is obtained than on fresh specimens. In a series of 81 specimens, negative after 15 minutes search, by the Phillip method, 8 were found positive after shaking 10 minutes and searching for 15 minutes. This is a very appreciable gain, and cannot be accounted for by the advantages offered in searching a second 15 minute period; for the second 15 minute period had been found to give only 3 positives in a total of 120 specimens negative for the first 15 minute period.

The Mechanical Shaker. The mechanical shaker breaks up the mucus in fresh specimens and distributes the bacilli, if present, uniformly through the specimen. It probably does not break up the bunches of bacilli appreciably within 10 or 15 minutes shaking. Differential counts of free bacilli and bunches after 20 and 40 minutes shaking show a slight resolution of bunches. On fermented specimens, I have compared the differential counts made after 5 minutes shaking, with those made after 20 minutes further shaking; the counts from the latter invariably show a smaller proportion of bacilli in bunches. The chief value of the shaker, however, is in securing the uniform distribution of bacilli and bunches throughout the specimens. A study of my records of rare bacilli shows that after shaking the occurrence of two free bacilli or bunches in the same field is an unusual finding, although, two or more may lie parallel cemented together by the interbacillary wax. This definite picture after shaking gave me opportunity recently to exclude the positive findings in a specimen as a contamination; I had searched for 4 minutes without result, and suddenly came to a single field containing 1 bunch of 20, 2 pairs of bacilli, 1 bunch of 3, and 17 free bacilli. The bunches and free bacilli did not touch each other at all. Search for the rest of the 15 minute period failed to give a single bacillus. Such a picture is common in direct smear, but in over 160 detailed records which I had made following the shaking method, I had never seen such poor distribution before. The findings were surely a contamination, probably from a droplet of bacillus laden material from another sputum which flew on the slide while sterilizing the loop. On identifying the specimen, it was found to come from a patient who had given a negative result at the previous examination, and in whom bacilli had never been found before. A new specimen was collected which gave a negative result. In this way I feel that the shaking method affords a splendid control over possible contamination of glassware and of preparations after they have been made. Chance contaminations must surely take place occasionally in all laboratories.

The shaking method has been modified variously. Thelinius shook fresh sputum with antiformin. Kinyoun proposes a technic which probably approaches nearer to the ideal than any that has yet been suggested. His routine method consists in adding 1 c.c. of ligroin to the specimen and an indefinite amount of alkaline hypochlorites. The mixture is then shaken 5 to 10 minutes in the shaker, and transferred to the centrifuge at low speed. The ligroin carries the detritus with the bacilli to the top, forming a soapy layer, from which the smear is made. The advantage in this method is that it combines the action of ligroin in reducing the bunches of bacilli into their component free cells with the thorough distribution attained by the hypochlorites and shaker.

The Ellermann and Erlandsen technic was developed as the result of the most painstaking inquiry into the physical characteristics of sputum and the *modus operandi* of the processes recommended, that has appeared in the literature. Their aim was concentration, rather than homogenization. The technic as proposed does not secure as thorough homogenization as is obtained with the shaker. They propose a modified fermentation method followed by the centrifuge. The method is: To 1 volume of sputum add ½ volume .6 per cent Na_2CO_3. Mix. Set at 37° for 24 hours. Pour off fluid. Centrifuge. Pour off again. Now add to 1 volume of sediment, 4 volumes .25 per cent NaOH. Stir thoroughly. Bring to a boil and centrifuge. The method gave three times as many bacilli in the same amount of sediment as did the Phillip method.

It is generally agreed that sodium hydrate in strong solution either destroys the tubercle bacilli or injures them so that they do not stain as deeply as when fresh. Even with the low percentage of sodium hydrate recommended, I have not been able to obtain as deeply stained bacilli by the Ellermann and Erlandsen method as are obtained in fresh smears or after simple fermentation of the same specimens. Preparations by the Ellermann and Erlandsen method sent me by other workers invariably show a marked diminution in stain penetration—far below my routine standard. The same is true of all the antiformin methods which I have tried. Another disadvantage in these methods is the concentration of dust particles which interferes materially with morphological studies. The dust cell found so commonly in the sputum remains intact after simple shaking, but if a solvent is used—sodium hydrate, hypochlorites, etc.—the cell is disintegrated and hundreds of dust particles are scattered through the specimen. This is a decided disadvantage.

The Wet Method of Making Preparations.—In making slide preparations, regardless of the technic in preparing the material, there is a universal tendency to transfer the sediment to be examined in as dry a condition as possible to the slide, and depend upon distributing the material uniformly with the loop. It is impossible to get preparations of uniform thickness in this way. The method which I use is to add one or more volumes of water or salt solution (or one per cent egg albumen, if working with a centrifuge residue), which, when thoroughly shaken by hand, may be poured out on the slide. In this way the particles settle evenly in drying. The slides placed on a constant temperature bath at 65° are tilted laterally in drying, so that the film will be thin on one side and thick on the other; an ideal section will be found between these extremes, for searching. Fermented specimens rarely peel off in preparation, unless they have fermented for several days. Sputa from lung abscess cases, because of the small amount of mucus and albumin present, are the only exception. It is always advisable to add egg albumen to washed centrifuged residue, as all mucus and albumin native to the sputum has been lost.

Staining of Tubercle Bacilli.—The Ziehl-Neelsen staining method is the one generally employed. The stain is made as follows: fuchsin, 1 gm.; absolute alcohol, 10 c.c.; 5 per cent carbolic acid, 100 c.c. The preparation is covered with the stain and heated over the flame until the boiling point is reached. Some advise boiling for three or four minutes, adding new stain as the first evaporates. Should the specimen become dry, it will be hard to decolorize and crystals of stain will be precipitated, which are confusing in the search for bacilli. Staining in the cold or at 37° for 24 hours has been recommended. Such specimens are, however, much more difficult to decolorize with weaker acids and alcohol. For decolorizing, a number of reagents may be used with a considerable range in strength. The tendency has been to use acids and alcohol as strong as may be, without injuring bacilli. Nitric acid 25 per cent, hydrochloric acid 2 per cent, either alone or in 80 per cent alcohol, and sulphuric acid 10 per cent in 95 per cent alcohol, are employed by various workers. If acid is used alone, the decolorizing must be completed with alcohol. For counterstaining, Loeffler's methylene blue is almost invariably used (30 c.c. saturated solution methylene blue in alcohol, 100 c.c. of a 1 to 10,000 aqueous potassium hydrate solution).

In my own practice I rarely use any of the above modifications. The acids in such strength and 95 per cent alcohol removes much of the color from the bacilli. I prefer maximum penetration and retention of stain in the bacilli, even though the criticism be raised that there is considerable likelihood that other acid-fast and alcohol-fast organisms occasionally will be present because of the weakness in strength of the decolorizing agents. I have followed Spengler's staining method rather closely. He heats only to a faint steam, which represents a temperature of 60° to 65° C.

The smear is covered with Loeffler's methylene blue ½ to ⅓ strength, allowed to remain for 30 seconds and excess poured off. As soon as dry, flood with carbol fuchsin and place on constant temperature water bath at 65°. Let stand 3 to 5 minutes. Pour off. Do not wash. Flood with Esbach's reagent; agitate for 10 seconds. Add 1 drop 15 per cent nitric acid; agitate 10 seconds. Pour off. Decolorize with 70 per cent alcohol. Wash with warm water between applications of alcohol if desired.

Counterstain weakly with Esbach's. Wash off excess reagent and dry.

The use of methylene blue is to stain the envelope of the bacillus so that the resultant color of the bacillus is not pure red as by Ziehl's method, but a deep purple. The use of the oven gives uniform heat to all specimens—a very important point if comparisons are to be made. Spengler used the first application of Esbach's as a mordant, stating that the bacilli were less likely to be decolorized. I have no opinion on this point, but it acts as a diluent for the nitric acid used, and in many specimens imparts sufficient color so that the final counterstaining is not necessary. The special features of the technic are the low heat applied, weak nitric acid (not over 1 to 2 per cent) and weak alcohol; and finally the substitution of a yellow background for the classical methylene blue. Any one who will employ this background long enough to accustom the eye to the change, will, I believe, never return to methylene blue. Yellow is a better contrast to red or purple than is blue. The pus and epithelial cells are scarcely seen, in marked contrast to the picture when methylene blue is used. Consequently all bacilli stand out at a glance and one does not hesitate to determine whether an artifact at the edge of a cell is a bacillus or not, as it is often necessary to do when methylene blue is used. One can search a much thicker field because yellow intercepts less light than blue, and thereby increase the chance of making a diagnosis in less time. I have used this technic in staining over 9,000 sputa in the last eight years, and employ it exclusively for routine work. It is a satisfaction to learn that others are slowly adopting the counterstain, at least.

With this method one secures a maximum penetration of stain in all specimens except in those admixed with food particles, as previously mentioned. The number of pale bacilli in smears of the same thickness average much less than where the older method is used. In making differential counts of bacilli to determine the index, I often classify them according to the intensity of stain as deeply stained, well stained, and pale. These observations are made in the thinnest parts, where little background is seen. Pale bacilli rarely exceed 5 per cent, and then they are usually found in bunches. In many specimens less than one per cent of pale bacilli are found in 500 studied. In practice I never make a diagnosis from pale bacilli, but insist on the presence of deeply stained and well stained types. This, I believe, anticipates the possible criticism that other acid-fast organisms might be included.

I have studied seven different strains of smegma prepared and stained as described, with the result that they were uniformly pale, and would therefore be excluded from consideration as tubercle bacilli.

Gram-Positive But Non-Acid-Fast-Forms of Bacilli.—The interesting observations of Much, from which he concluded that there is another well defined form of the tubercle bacillus which does not retain the stain after decolorizing with acid and alcohol, promised to throw new light upon that group of patients who never show acid-fast bacilli, but yet present all the clinical symptoms of tuberculosis. Guinea pig inoculations often result positively in these cases, yet no bacilli are found by the usual stain. Much found that the organisms in these cases were Gram-positive; but that they had apparently lost their envelope, which by the Ziehl staining method, retains the stain.

Preparations of lymph glands and other tissues, in which he was unable to demonstrate the acid-fast forms, were injected into pigs, producing tuberculosis; he was able to recover the acid-fast tubercle bacillus from the diseased organs of the animals. Material from a cold abscess in which acid-fast forms had not been found, produced typical lesions in pigs from which he recovered acid-fast forms. Thirty preparations of the material were stained by Ziehl and the rapid Gram method without result; but in all cell remnants were demonstrated fine granula after staining 24 hours at 37° with methyl violet.

In culture tubes the only growth up to the eighth day was of the Gram-positive type; thereafter acid-fast rods were found, showing the transformation of the former into the latter. The stained picture is that of several fine granules in linear arrangement, indicating that they are joined together.

The stains recommended are:

Gram	I.	Analine oil—gentian violet	5 min.
		Lugol solution	1 min.
		Decolorize in absolute alcohol and clove oil.	

Gram II. Methyl violet B. N. 10 *c.c.* saturated alcoholic solution in 100 *c.c.*
2% carbolic acid (heat over flame, or 24-48 hrs. at 37°).
Potass. iodide—iodine solution 1-5 min. 3% hydrochlorine acid
10 sec.; finally with equal parts acetone and alcohol.

Gram III. Methyl violet B. N. as above.
Potassium iodide—H_2O_2 (5 gm. K I; 100 cm. 2% H_2O_2 2 min.)
Decolorize with absolute alcohol.

In practice the difficulties in differentiating the Gram-positive forms from other organisms seems to be the chief difficulty in the method. If using the method for the examination of sputum, fresh specimens only may be used before fermentation with growth of other organisms, has taken place.

Indirect Methods for the Demonstration of Bacilli.—In case bacilli are negative in sputum, pig inoculation may be tried. Suspected sputum may be shaken with a one per cent solution of sodium hydrate to demucinize; or treated with 10 to 20 per cent antiformin and an emulsion made from the centrifuged product. I have used untreated sputum to a considerable extent. By quickly heating to 55 degrees, the viscosity is markedly lowered, and the fresh material will pass through a large needle slowly with sufficient pressure. Injection behind the shoulder, in the inguinal region or into the peritoneal cavity is the usual procedure. It has been recommended to crush the lymphatic glands before injecting, if the shoulder or inguinal region is chosen. In this way injured glands are supposed to be more likely to become infected. Intraperitoneal inoculation is probably used the most.

The pig should be weighed before inoculation, given hygienic quarters, and weighed again at death or at the time of killing, which is two to three weeks.

While not proposed for diagnostic purposes, Petroff's method of sputum culture offers a possible substitute for pig inoculation. The culture medium consists of 1 part whole egg, 1 part of meat juice, and gentian violet 1 to 10,000. It is based on the fact that gentian violet inhibits the growth of all organisms found in sputum except the tubercle bacillus. Infuse 500 grams of beef in 500 c.c. of 15 per cent glycerine in water. Squeeze in sterile press 24 hours later. Collect in sterile beaker. Sterilize eggs in 70 per cent alcohol or hot water. Break in beaker.

Mix thoroughly. Filter through sterile gauze. Add 1 part of meat juice. Add sufficient gentian violet to make 1 to 10,000. Tube in 3 c.c. quantities; inspissate for 3 successive days; 1st day at 85° until solidified; 2nd and 3rd days at 75° for 1 hour.

The sputum is shaken with 3 per cent sodium hydrate and placed at 37° for 20-30 minutes; then neutralized to sterile litmus paper with normal HCl. Centrifuge and inoculate residue. Petroff obtained cultures in 69 cases without a single contamination. Six of these positives were from patients who had never shown bacilli before.

Number of Bacilli.—The importance of the relative number of bacilli in sputum has been discussed by many clinicians, especially as to its value in prognosis. Various detailed methods have been suggested for reporting the findings, the best known of which is Gaffky's scale. In view of the fact that the older technics did not secure homogenization of bacilli, it is not probable that any very definite information has been obtained by this method which is of value in observing the individual patient. Most of the estimates have been made from single specimens without any effort at thorough homogenization. In a general way, however, all observers agree that the greater the relative number of bacilli, the severer the process and the graver the prognosis.

Nuttall showed years ago that the total number of bacilli, as determined by exact methods, does not vary greatly from day to day in the same patient. I made daily counts of bacilli from one patient with chronic tuberculosis with but few symptoms, for a period of eleven days. The total bacilli in the 24 hour specimens for the successive days were as follows: 128. 130, 133, 83, 98, 131, 97, 118, 56, 103, and 117, expressed in millions of bacilli. With the exception of the number for the 9th day, 56 millions, the totals are remarkably close.

Individual exact counts were made on 24 hour specimens from forty-three different patients, the counts ranging from one hundred and forty thousand to one billion in cases of recent cavity formation and of moderately extensive involvement, and from one billion to ten billions in those with massive involvement; one count gave thirty billions. These counts were sufficient in number to confirm the earlier work of others, and to show that considerable importance may be attached to bacillary estimation provided that the error be not too great. It is of course impracticable to make exact counts as a matter of routine because of the time consumed.

My method of estimating the number of bacilli is as follows: Twenty-four hour specimens of sputum are homogenized 10 to 20 minutes in the mechanical shaker, either fresh or after fermentation. The bacilli are by this means thoroughly distributed. The number of deeply stained bacilli per field is estimated. Well stained and pale bacilli are ignored. If less than one per field, say 1 bacillus in 5 fields, the number recorded is $\frac{1}{5}$. If one or more per field, the record is 1, 2, 3, 5, 10, 20, etc. Now it is evident that the number of bacilli per field depends upon the relative proportion of solids in the sputum. If we assume a constant total number of bacilli, the number of bacilli per field will decrease as the amount of solids is increased; or the number per field will increase as the amount of solids is decreased. In making this statement we ignore the albumin and mucus content, since the former is decanted after fermentation and the latter is destroyed in that process. The relative number of bacilli in 24 hour specimens, therefore, may be determined by multiplying the estimated number per field by the number of c.c. in the sediment volume. This computation corrects the well known fallacy where only the number per field is estimated.

Although the error in estimating the number of bacilli per field is considerable, such error is allowable when we consider the great variation in the actual numbers of bacilli from different patients; and there is still a good opportunity to classify patients based upon these estimates. The error should not usually exceed 100 per cent.

The demonstration of the tubercle bacillus in sputum, feces, or urine, remains the most important fact in diagnosis. There are, however, certain theoretical objections to accepting all positive findings of bacilli, particularly in sputum, as indicative of tuberculosis. Here arises the question of the possibility of contamination of the secretions by bacillus-laden dust, particularly in cities. I was not impressed with this possibility until I had determined by accurate counting methods the numbers of bacilli found in 24 hour specimens from patients, and the numbers found in smears from the same cases within a definite time of search. In one count, 280,000 bacilli were computed for the total specimen in which the sediment volume was 5 c.c. In stained preparations only 10 bacilli were found in 18 minutes' search.

Now, if we suppose that the number of bacilli and the sediment volume were correspondingly reduced, say ten-fold, we would have 28,000 bacilli in ½ c.c. of sediment volume, with the same results microscopically. Further we might reduce the microscopic finding to 1 or 2 bacilli in 18 minutes' search, which would indicate the presence of only 2,800 and 5,600 bacilli respectively. As many 24 hour sputa contain less than ½ c.c. of sediment volume, 1 or 2 bacilli might be found in 18 minutes, although only 500 to 1,000 bacilli were present in the whole specimen.

As to the possibility of contamination from bacillus-laden dust, in the above example we have but to consider the following record: 24 hour quantity, 45 c.c.; sediment volume, 8 c.c.; total bacilli, 3¼ billion; bacilli per field, 30. This report was from a patient with chronic tuberculosis, without temperature, and walking about. There are thousands of persons presenting this picture in every large city, part of whom at least are expectorating on the streets. Now, if the 8 c.c. representing the sediment volume above were dried, it would weigh about one gram; and if distributed into one million particles, as it probably would be by the action of the wind and passing vehicles if expectorated on the street, each particle would contain on an average 3,250 bacilli, and would weigh only 1/1000 of a milligram.

Bacilli of such small number, lodging in the nasal and bronchial secretion, would be demonstrated by our modern methods of sputum examination. In my practice, bacilli occurring in the sputum of unknown cases in number less than 5 bacilli in 15 minutes search are considered doubtful for diagnostic purposes. If such findings are found in a second specimen, I consider the diagnosis made.

It does not seem that the results from pig inoculation, whether positive or negative, conclusively prove the point, though it is to be expected that many if not all of such bacilli would be dead, due to the action of sunlight. On the other hand, pigs have been infected with as small a number as ten bacilli, which rather indicates that they are too sensitive and may develop the disease from contaminated material.

For diagnostic purposes, the interpretation of positive findings obtained by the cultural method of Petroff must be made with still greater reservation. In fact, it is quite probable that there is a practical limit to the sensitiveness of technics for the demonstration of tubercle bacilli for diagnostic purposes, at least in sputum, urine, or feces where opportunity is offered for contamination; and if the more sensitive methods are used, it must be with the knowledge of added responsibility in interpretation of results, if found positive.

Morphological Classification of Tubercle Bacilli.—For eight years I have recorded the following general description of the bacilli found in smear preparations: Number per field; whether extra long, medium or short; extra broad, broad or narrow; uniformly stained or beaded; deeply stained, well stained or pale; bunched or free. By employing the initial letter of each descriptive term a complete description is recorded with a minimum of writing. The three mathematical signs, =, <, ≪, indicate the relative proportions of the characteristics present. = is used where one of two characteristics is from 40 to 60 per cent of the other; < means more than or less than, and denotes that the lesser characteristic is from 20 per cent to 40 per cent of the greater; and ≪ means that the lesser is from 10 per cent to 20 per cent of the greater. Less than 10 per cent is not recorded. The following illustrates the method of recording:

50 per field 1 + < 1 < m; br – n; b > u – s; d – s < w – s; bun > free.

This form of report, while not very accurate for the individual case, gives considerable information in the aggregate. The difficulty in interpreting such findings is due to the uncertainties incident to our homogenizing methods, variable penetration of stain and to the well known difficulty of securing smears of uniform thickness. For homogenizing, the mechanical shaker is indispensable. As to penetration of stain, there is considerable variation with which bacilli from different sputa are impregnated. This is often noticed when two preparations are placed on the same slide and stained together. In one, the bacilli stain intensely and uniformly; in the other they are only well stained or pale. The latter, if washed with ether, on restaining will often take the stain as intensely as the other specimen does. In such sputa, after fermentation one will usually find considerable neutral fat floating on the top of the supernatant liquid. This seems to account for much of the defective staining, which is likely to be attributed to inherent differences in bacilli. This fat is usually easily traceable to food particles,—butter, milk, etc., where the patient is careless about cleansing the mouth after eating.

Even after all other technical features have been carefully carried out, such as thorough homogenization, uniform temperature during staining, definite time for exposure to stain, etc., one may obtain quite different descriptions from the same specimen unless very careful to choose fields of the same thickness. Bacilli which appear narrow, beaded, well-stained, or pale in thick fields, are broad and deeply stained in very thin fields, and the beads may not be seen at all, or only slightly visible, being masked by the intense staining of the bacillary envelopes.

The morphological characteristic which is most free from error in recording is the relative length of the bacilli. From 50 to 80 per cent of all tubercle bacilli in all cases are from 2 to 4 microns in length; the remaining 50 to 20 per cent varying greatly in length. All may be less than 2 μ. or they may be from 4 to 10 μ. in length; consequently, the average length of bacilli met with in different sputa shows striking variations.

During the last year I have added to the above form of report a fairly accurate classification of bacilli based upon the length, from which is calculated an index in the following manner. Into the ocular is inserted a little brass frame upon which is mounted three glass beads which cast shadows of 2, 4, and 6 microns upon the microscopic field, using Bausch and Lomb eyepiece 1 inch or 10x, objective $\frac{1}{12}$th, focal distance 160 mm. (see Fig. 100). These glass beads are made by drawing out very fine glass filaments in the flame. They are then fused on the end in a very small flame, preferably from an alcohol burner. They are then standardized with the stage and eye-piece micrometers, and those selected which are of the desired diameter. Unfortunately the manufacturers do not make their oil immersion lenses exactly of the same magnification, so that one cannot use the device in different microscopes unless the ocular and objective systems are exactly the same. I have three B & L $\frac{1}{12}$ oil immersion lenses, and the relative magnifying powers are 79.2, 75, and 71.4. This results in a variation of about 10 points in index in comparisons which I have made, between the first and last objectives. There would be a great advantage if the method could be carefully standarized so that a given index would mean the same regardless of the instrument used.

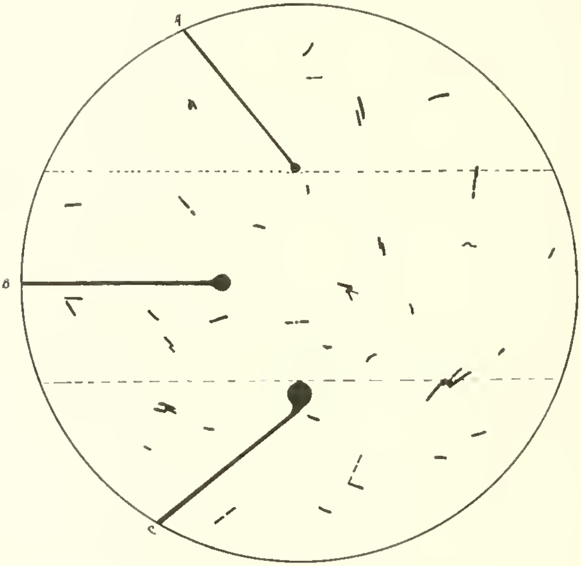

Fig. 100.—Device for classifying tubercle bacilli according to length. *A, B,* and *C,* fine glass filaments supporting glass beads which cast shadows of 2, 4 and 6 microns respectively, upon the microscopic field.

Because of the ease with which the device may be broken, I have attempted to find some substitute. A special disc was made by the manufacturer upon which were made three circular etchings of the proper size; but it was necessary to discard the device because the etchings did not show up plainly. With a little patience I believe that anyone familiar with micrometry can manufacture the device for himself. I have used mine for fifteen months without breaking.

Using a mechanical stage all bacilli that pass between beads 2 μ. and 6 μ. are classified. This can be done quite rapidly, and in most preparations it is not necessary to orientate the beads, a glance being sufficient to determine whether a given bacillus is longer or shorter than the diameter of a certain bead. There are some preparations, however, that give considerable trouble, in which the greater number of bacilli are so near 2 μ. and 6 μ. in length that one is in doubt unless the bacillus is brought alongside the bead. Very thin smears only should be used. The bacilli must lie absolutely flat and not tilted as we find in thick fields. Even then there is some inaccuracy. A differential count of 500 should be made if possible. This reduces the error to a small figure.

Length Index of Tubercle Bacilli.—The index is derived from the differential in the following manner: All bacilli less than 2 μ. in length are called short and are given an arbitrary value of 1. All between 2 μ. and 4 μ. in length are called medium. An arbitrary value of 2 is assigned. All between 4 μ. and 6 μ. are called long, with a value of 3. All longer than 6 μ. are called extra long with a value of 4. A count of 100 is the basis.

	4	3	2	1	
Differential Counts.	Extra-long	long	medium	short	Index
	8	27	72	3	260
		1	64	35	166
			39	61	139

The index determined in this way is called the *length index of tubercle bacilli*. In over 600 differentials made the index varies from 130 to 264,—about 75 per cent of them lying between 150 and 200. The index cannot be made safely from single specimens, nor from unhomogenized 24 hour specimens, as is shown by the following experiment: A patient with signs of considerable activity was instructed to separate his 24 hour sputum into three receptacles. He raised 35 c.c. from 8:00 P. M. to 6:00 A. M.; 55 c.c. from 6:00 A. M. to 9:00 A. M., and 75 c.c. from 9:00 A. M. to 8:00 P. M. Two direct smears were made from each of the three portions, and the index determined from each.

The six indices were 180, 201; 185, 190; 187, 190. Although four of the indices accord splendidly, a variation of 180 to 201 cannot be allowed. The experiment has been repeated with other patients with essentially the same result.

Comparisons after shaking 10 to 20 minutes show less variation between duplicate counts. Two series of duplicate counts on different sputa gave the following indices: 214, 215; 165, 158. Several series of counts of four or five preparations from each specimen gave 156, 155, 161, 160, 153; 202, 208, 210, 217, 214; 223, 223, 221, 218. Although the index made from homogenized specimens is usually more accurate on the average than when made from unhomogenized material, at least 5 or 6 points should be allowed for error.

URINE.

The value of routine examination of urine in the study of tuberculous patients is not appreciated as fully as it should be. The impression is too general that except in special instances, where some non-tuberculous complication is suspected, urine analysis offers little help to the clinician and is hardly worth the time required. Such records as are made are usually determined from single specimens which may give quite different information from what would be obtained if twenty-four hour specimens were analyzed.

For eight years I have made an analysis of a twenty-four hour specimen from each patient previous to the regular monthly physical examination. This analysis consists in the determination of the specific gravity, the presence or absence of serum albumin, globulin, nucleo-albumin, mucus, reducing bodies, indican, and diazo. For the last two years the urochromogen test has also been made regularly. Formerly quantitative chloride and phosphate determinations were made, but later were discontinued as regular tests. Over 8,800 such analyses have been made. The tests which have been most useful are the diazo and indican tests.

Collection of Specimen.—Formerly the twenty-four hour quantity was collected from morning to morning according to the well known directions, and, although the instructions were carefully given, there was often confusion in the patient's mind and errors were common; so that the time for starting and ending was changed to eight o'clock in the evening, since at that hour any possible error in voiding more or less than the proper amount would be less than in the case of a similar error in voiding the more concentrated morning's urine.

There is no great objection in allowing the urine to stand over night, as the substances tested for are relatively stable. Traces of albumin may, however, be formed by bacterial autolysis, hence the presence of albumin must be interpreted with this fact in mind. A few drops of chloroform are added to limit bacterial growth.

The Diazo Reaction.—The diazo test is made with the usual reagents.

Sol. I. One-half per cent sodium nitrite.

Sol. II.	Sulphanilic acid	5 gm.
	Hydrochloric acid	50 c.c.
	Distilled water to	1000 c.c.

In making the test, one-tenth c.c. of Sol. I is mixed with each 5 c.c. of Sol. II. About 2 c.c. of urine is placed in a test tube one-half inch in diameter, and to it is added an equal amount of the reagent. The mixture is shaken briskly to develop a good foam. One-tenth volume of ammonia is now added and a pink color in the foam is looked for. The pink is often masked by an admixture of brown. In such cases doubt may be cleared by adding only one-half as much reagent as urine. In a series of tests on 48 consecutive urines in which 21 positive and 10 doubtful reactions were obtained by using equal volumes of reagent and urine, sufficient additional results were obtained by using ½ volume reagent to 1 volume of urine to make 29 positive and 6 doubtful reactions in all. While the additional advantage given by the latter proportions of reagent and urine has long been known, it is undoubtedly overlooked in many laboratories.

When the diazo is positive, the relative intensity is determined by diluting the urine and making the test again, always with equal parts of the reagent and diluted urine. The dilution at which the pink is just visible in the foam is recorded as the degree of intensity. One must be very exact in making the dilution and in measuring the urine and reagent, otherwise the results may be inaccurate. In over 2,000 positive tests found in over 8,800 urines from 1,500 patients, diazos were found commonly ranging from a doubtful reaction to one-sixth, rarely to one-seventh, one-eighth, or one-ninth. As the diazo is usually more intense in the afternoon than in the morning, positive tests have been found at that time in one-tenth and one-twelfth dilutions.

Although the diazo reaction has been known for over thirty years, individuals will differ somewhat in deciding whether a given test is positive or negative. The pink foam is probably the most generally recognized criterion. Normal urines give a white foam if fresh, or a very light brown foam if the urine has stood for some time, or is concentrated. The solution below the foam ranges from light yellow to dark yellow, sometimes with a slight brownish tone, but never in my experience does it assume a reddish tone in a normal urine. When doubt exists as to the presence of a pink in the foam, I call the reaction a *suggestion.* Patients showing such doubtful reactions in the twenty-four hour urine will nearly always show a distinct diazo if an afternoon sample is called for. These doubtful reactions always show a reddish tone in the solution, although it may be partially masked by admixture of brown.

The Urochromogen Reaction.—In announcing his permanganate test for the urochromogen body, Weisz considered the relative proportions of reagents and urine to be of little consequence. This fact gave it great advantage in his mind over the diazo test, in which care is required to use exact amounts of reagent and urine. He recommended to dilute the urine roughly with two parts of water in a test tube, and add three drops of potassium permanganate. The development of a yellow color was considered a positive test. A number of observers following these instructions have arrived at widely different conclusions as to the relative sensitiveness of the urochromogen and diazo tests, and consequently as to their interpretation for purposes of diagnosis and particularly prognosis in tuberculosis. All reports agree, however, that the urochromogen is met more frequently than is the diazo. Heflebower, Gullbring, Sinclair, and others found it from two to three times as often as the diazo, but

Schaeffle found it in only ¼ more cases than he did the diazo. Others found the relative occurrence of the two reactions lying between these extremes.

From the simple instructions of Weisz, it is evident that two workers may make their tests with widely different proportions of reagent and urine, depending upon the size of drop of reagent and the amount of urine used. As it seemed that this might account for some of the differences shown in the reports, I undertook a detailed study of both urochromogen and diazo in a considerable number of fresh urines from normal persons and patients, and in twenty-four hour specimens from patients. The color of both foam and solution in the diazo was described. The urochromogen tests were made in a series of five tubes, each containing 5 c.c. of ⅓ dilution of urine. Using one tube as a control, to the four tubes remaining were added successively, quantities of reagent corresponding to 5 drops, 3 drops, 2 drops and 1 drop,—this being the probable range of variation employed by various workers. The changes of color were recorded in each tube at once, at the end of 15 seconds, of 30 seconds, 60 seconds, and 120 seconds. If either the diazo or urochromogen was positive on first trial, the urine was further diluted and the test repeated until the dilution was found in which the pink disappeared in the foam and solution of the diazo test, and the yellow disappeared in the urochromogen test.

From this work it was found that yellow reactions indistinguishable in color from the true urochromogen reactions occurred in the majority of fifty-eight normal urines. These reactions were mostly transient and their permanency depended directly upon the amount of reagent used. Thus, in a very small number of urines, the yellow persisted beyond 30 seconds in the 5 and 3 drop tubes, and beyond 15 seconds in the 2 drop tube, while all yellow had disappeared in the 1 drop tube at 15 seconds. For this reason the time limit for transient reactions was established at 60 seconds for the 5 and 3 drop tubes, at 30 seconds for the 2 drop tube, and 15 seconds for the 1 drop tube. Yellow reactions persisting beyond these time limits were interpreted as positive reactions. In the two series of 94 fresh morning urines and 167 twenty-four hour specimens from patients in which no red was found in the diazo solution, there was a slight tendency for the yellow to persist in the 2 drop and 1 drop tubes beyond their respective time limits for normal urines. In fact, 1 positive reaction was found in the 2 drop tube, and 2 positive reactions were found in the 1 drop tube, while the 5 and 3 drop tubes were negative. This clearly indicated that the lesser quantities of reagent were more sensitive in demonstrating very slight reactions than were the larger quantities of reagent. In four of the above urines, showing a slight diazo reaction, the urochromogen was recorded negative in the ⅓ dilution of urine, in all tubes. Diluting the urine to ⅙ and repeating the test gave distinct reactions in the 2 and 1 drop tubes, in two of the urines, and questionable reactions in the other two. All these urines were deep yellow in color. The influence of the age of the specimen on the reactions was determined by comparing the reactions obtained on fresh normal specimens, with the reactions obtained twenty-four hours later, on the same specimens. The difference was insignificant, showing a slight loss in the permanency of the yellow.

From these facts, the following standardization of Weisz's urochromogen test was attempted. Place 5 c.c. of ⅓ dilution of urine (1 part u. and 2 parts water) into each of two test tubes, selected so that the column of liquid is 3.5 cm. deep. Hold both tubes over a white background, which reflects the light strongly and place 0.1 c.c. potassium permanganate (1:1000) in one of them. Shake quickly and look into the tubes from above. Record any increase of yellow at the end of 30 seconds as a positive reaction. If in doubt repeat the test, using only .05 c.c. of reagent and look for yellow at 15 seconds. If still in doubt use a ⅕ or ⅙ dilution of urine, and repeat the test.

Indican Determination.—For the determination of indican I have employed the reagents recommended by Robin, adding a quantitative modification which may be carried out quite accurately.

To 20 c.c. of urine add 2 c.c. sat. sol. lead acetate. Filter. To 10 c.c. filtrate add 10 c.c. Obermayer's reagent. Set aside to cool about 30 minutes. Add 20 drops of chloroform. Shake 30 seconds. Let stand 2 minutes. Add potassium chlorate solution (34.6 gm.:1000 H_2O) drop by drop, shaking twice as above, between drops until no blue remains in the chloroform. One soon learns to judge as to the approximate number of drops required in a given specimen, so that in a chloroform extract requiring 15 drops to decolorize, nine or ten drops may be safely added at once. Five minutes should be allowed each time for the full effect of the previous drops, before new quantities of the reagent are added.

To carry out the test conveniently and accurately, I have used a small bulb bottle blown from glass tubing, of about 30 c.c. capacity, pouring the filtrate into it from a graduate and then rinsing the graduate with the Obermayer's reagent. The drops are added by means of a standardized pipette, the outer surface above the point of which is covered with paraffine. The $KClO_3$ thus adheres only to the end. Fifty drops equals exactly 1 c.c. if dropped slowly to avoid forcing off the drop by rapid currents. With care the paraffine need not be replaced for two or three weeks, although if the drop adheres in the least to the wall above the point, it will be too large. I have tested this dropping method day after day for two weeks, under diverse

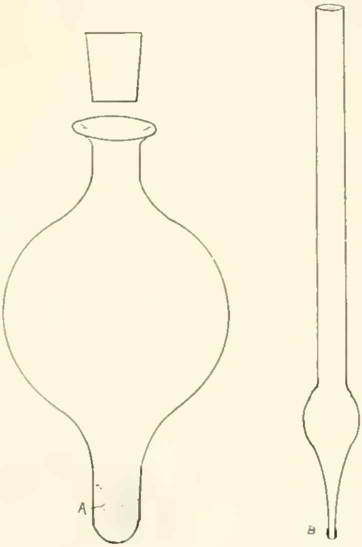

Fig. 101.—Bulb and pipette for convenience in making quantitative indican determinations. *A*, chloroform extract in bottom of bulb; *B*, paraffine on outer wall of point of pipette.

atmospheric conditions and have found that the variation was less than 1 drop from 50 drops to the c.c. (See Fig. 101.)

The result of a given test is expressed in the number of c.c. of $KClO_3$ which it would take to decolorize the total twenty-four hour quantity of indican by the following formula.

$$\frac{\text{Number of drop}}{50} \times 10 \times \text{Number of hundreds of urine.}$$

Thus, if in a urine of 1500 c.c. it takes 6 drops to decolorize the indican in 10 c.c. of filtrate, the application of the formula would be

$$\frac{6 \times 10 \times 15}{50} = 18.$$

The average elimination of indican in twenty-seven normal persons on a mixed diet was found to be fifteen by this method. The color and clearness or cloudiness of the chloroform extract should be noted. In normal persons the bleached extract ranges from no color at all to light shades of brown, or yellow or mixtures of both, and is perfectly clear. With stomach and bowel disturbances the extract gives deeper shades usually with the addition of red, though sometimes dull green shades are found. The extract is cloudy in the majority of such specimens.

The other tests mentioned, require little notice as they are well known. In employing Fehling's solution for reducing bodies, I have been accustomed to indicate the slight degree of reduction by substances other than glucose. The degree of reduction is indicated by the sign + meaning a blue green, + + a pure green, and + + + a yellow or colorless reaction. These reactions are recorded at the end of two minutes.

Tubercle Bacilli in Urine.—Cases of tuberculosis of the kidney or bladder rarely come to us; so that no effort has been made to study the urine for tubercle bacilli, except where pus is present. I invariably examine for bacilli in such cases. The technic employed is the use of the mechanical shaker followed by the addition of .25 per cent sodium hydrate according to the Ellermann and Erlandsen technic, if concentration is desired.

The bunching of bacilli in urine is extreme as compared with the findings in sputum. I have made differential counts of free bacilli and bunches in eleven different specimens from as many patients. The largest ratio of total bacilli to the number of free bacilli and individual bunches was 11.904; that is, with complete resolution of bunches and homogenization, the chance of finding the first bacillus in a given period of time would be increased 1090.4 per cent. The average ratio of the 11 specimens was 3.597, showing a much greater tendency to bunching than is observed in bacilli in sputum (see Tables VI and VII). In one case I had an opportunity to compare the ratios found in the urine specimen and in caseous material from the kidney after nephrectomy. The ratios from urine and kidney were respectively 2.833 and 2.718. The cavity containing this material did not communicate with the pelvis of the kidney.

From these observations, it might be suggested that for diagnostic purposes, such technic is indicated as secures resolution of bunches and thorough homogenization. This is found in the mechanical shaker after 5 per cent xylol or ligroin has been added to the specimen. In one specimen in which 5 per cent xylol was added and shaken 20 minutes almost perfect resolution was attained. The direct smear gave a differential of 1000 free bacilli to 4223 in bunches; shaking with xylol gave 1000 free bacilli to 58 in bunches. On the other hand, the bunching is so characteristic of tubercle bacilli, that one wonders if it is not sufficient in itself to determine a positive diagnosis, and therefore should not be disturbed. Smegma bacilli, in my experience, do not show this arrangement. Because of my limited experience in these cases, I hesitate to recommend the xylol or ligroin method even though their efficiency in resolution is almost perfect.

BLOOD.

Although a large amount of work has been reported dealing with blood counts, hemoglobin estimation, differential counts, etc., the opinion is more or less prevalent that the results obtained do not justify the time required to make such observations, at least as a routine procedure. While agreeing with this view in a general way, I have, nevertheless, made these determinations as often as time would permit, hoping that we might be able to place a better interpretation upon them.

Practically all of my counts have been made within thirty minutes prior to the midday meal. The white cell count made at this time is from one-third to one-fourth greater than after the night's rest before the patient has stirred about.

The technic is the usual one, though instead of using a steel lancet, the edges of which are always rough, at least microscopically, a capillary glass tube is used, the end of which is sharpened into an angular point by inserting a small pointed instrument and prying first to one side and then to the other. The glass broken off leaves a lance point which is sharp and the edge is microscopically smooth. One can make a variety of cutting edges,—broad, narrow, long or short, and select the one which the conditions of the skin and the appearance of the surface circulation suggest. This method of puncture is far less painful than the old method, and the objectionable pressure on the part is no more necessary than with the usual method.

As the hemoglobin and red cell counts in the blood of the tuberculous give the picture of a simple anemia, I have not made the red cell count unless the anemia was severe. The hemoglobin is determined by the Sahli instrument. The white cell count, the general differential, Arneth's differential, and certain toxic changes in the neutrophiles, have been considered of most importance.

General Differential.—For the purpose of making differential counts No. 1 cover slips are used. In well made smears one may always study the error in distribution, while, with the slide method, there is an unknown amount of displacement of the larger cells, mainly transitionals, to the edge. There is some displacement with cover slips, but as the films are much more uniform in thickness than can be obtained by the slide method, the error can be corrected. The first hundred cells counted at the thick edge often give 10 to 15 per cent of transitionals, while the first hundred cells counted at the apex of the film gives from 0 to 5 per cent. So, in counting, I invariably select the middle third, avoiding both extremes. Hasting's and Giemsa's stains are used, preferably the former. Five hundred cells are always counted, classified as neutrophiles, basophiles, eosinophiles, large mononuclear, transitional and stimulation cells.

Arneth's Classification of Neutrophiles.—There is considerable difficulty in making smears suitable for Arneth's classification. Even in the best of films where there are from one to three thousand leucocytes suitable for a general differential count, one may, with difficulty, find three hundred neutrophiles satisfactory for Arneth's classification. Only the thin portions of the film may be counted lying between the center, where the cells are more or less crushed, and the outer thicker margin. The cells must be spread sufficiently so that the lobes of the nuclei may be readily recognized. However, in the best of films, there are 10 to 15 per cent of neutrophiles that defy accurate classification. I always rely on the number of connecting threads in case the lobes of the nuclei overlap, since there is always one thread less than there are lobes. I have not obtained as low indices on normal persons, as have been reported by some observers. I am inclined to believe that in some of these reports nuclear buds have been mistaken for lobes, and thus the index is lower than it should be. These buds are common in neutrophiles from the blood of the tuberculous as well as from the blood of apparently normal persons. They are connected with the parent lobes by threads indistinguishable from the threads connecting the lobes themselves, except that they spring from the side of the lobes, never from the ends, or long axis. I have counted as high as 25 of these buds in 100 neutrophiles, which would greatly lower the index, in case they were mistaken for lobes. Giemsa's stain is somewhat superior to Hasting's for Arneth's counts, but as it is less suitable for general differential counts, I have not used it to as great an extent. My average normal indices, as computed by the method of Bushnell and Treuholz is 57; that is, the sum of Classes I and II, plus ½ of Class III.

Nuclear and Protoplasmic Changes in the Neutrophile.—Years ago Holmes observed certain changes in the neutrophile of the tuberculous patient. My observations confirm in part, what he described, but there are some characteristics which appear of especial importance. These changes are always associated with a toxic condition, manifested by temperature and other signs of an active process. They consist in the appearance of small granular bodies in the protoplasm, approaching black in color. The prominence of these granules is designated by the signs |+, +, + +, and + + +, the first indicating very slight, and the last an extreme change, where the granules are large. The latter is invariably found in patients just before death. Coincident with this, the pink tone of the neutrophile tends to disappear, and the protoplasm approaches the basophilic type. I cannot say whether these dark, almost black granules are peculiar to tuberculosis, as I have not had sufficient experience in studying bloods in non-tuberculous infections. But records have been made of certain differences met with, in a case of appendicitis, one of typhoid fever, and a case of pernicious anemia. In none of these would the granules be confused with those present in the toxemia of tuberculosis. The case of appendicitis later came to operation and a simple catarrhal condition was found. The case of typhoid ran the usual course. This protoplasmic transformation or stippling, is never present with a normal Arneth count. But a marked Arneth change may occur without the protoplasmic change. As young cells always show greater affinity for aniline dyes than do the adult cells, some change would be expected in neutrophiles from patients with a high Arneth index, where the young cells are increased in the circulation. The stippling is always associated with a considerable degree of toxemia, with more or less constitutional symptoms, while a high Arneth index may be found in cases without such symptoms.

FECES.

Aside from the search for tubercle bacilli, the examination of the feces from tuberculous patients gives little or no information peculiar to the disease; consequently, routine examinations have not been made except where indicated by symptoms pointing to bowel complications.

Tubercle Bacilli.—In examining for tubercle bacilli, visible particles of mucus, if present, are homogenized, as in making direct smears of fresh sputum. In case none are found the specimen, after grinding in a mortar, is diluted with two parts distilled water, transferred to a bottle and shaken in the mechanical shaker. After allowing the coarse undigested particles to settle, a smear is made from the upper more finely suspended material. The preparation of the smear and staining are carried out as described under sputum, except that absolute alcohol is substituted for 70 per cent alcohol in decolorizing.

In 1907 and 1908 a study was made to determine what value the routine examination of feces would be to the clinician. The technic employed was as described above. The time of search, however, at that time, had not been standardized, for either sputum or feces, so only a guess may be ventured that it was 5 to 10 minutes.

In order to exclude bacilli from the lung as far as possible, the patient on entrance was cautioned about swallowing his sputum, and 5 days were allowed for previously swallowed bacilli to pass from the bowel. At the end of this time the specimen was taken. Two hundred and sixty-nine specimens were examined from 146 patients. Bacilli were present 107 times and absent 40 times in the feces of 68 patients with bacilli in the sputum, and they were present 4 times and absent 118 times in 78 patients without bacilli in the sputum.

Two types of other acid-fast organism were occasionally found,—one rather short, very deeply stained, and broader than the tubercle bacillus; the other very pale, and of rather close relationship morphologically to the tubercle bacillus. The first type would not be likely to confuse one who is familiar with the tubercle bacillus in sputum. The latter were distinguished by their uniformly weak acid-fast property; and I did not consider them tubercle bacilli. My later experience justifies this conclusion. On the whole, I do not consider that the differentiation of tubercle bacilli from other acid-fast organisms met with in the feces presents any very difficult problem, if care be taken to secure uniform penetration of stain. This point has been discussed under sputum.

The presence of tubercle bacilli in the feces seldom points to tuberculosis of the bowels, at least clinically. It is highly improbable that patients can guard against swallowing some of their sputum. As shown in the work above, in which bacilli were recovered in 73 per cent of all specimens from patients who showed bacilli in the sputum, only three of these patients gave evidence of intestinal tuberculosis.

It is sometimes strongly advocated to examine the feces from all suspected cases where sputum cannot be obtained. The four diagnoses made from examination of feces in my series apparently confirm this view, since in two of the four cases bacilli were later found in the sputum. But had the same care been taken in the collection of sputum at that time as is taken now, it is probable that those diagnoses would have been made from the sputum. In fact, since that time I have not found bacilli in the feces in a single instance, in which they were not also present in the sputum.

There have been three instances within the past five years, in which patients with clinically active lesions insisted that they had no sputum on first examination; but at succeeding examinations plenty of sputum with bacilli, was obtained. One of the three intentionally deceived us and the other two had not been properly instructed. Examination of feces in these cases might have given positive results. On the whole, however, the routine examination of feces for diagnostic purposes will be found a time-consuming procedure and a very poor substitute for thorough sputum examination.

It is sometimes necessary to examine the feces for occult blood. I have used the aloin and guaiac tests for this purpose. The feces are ground in a mortar; a small portion (1-2 gm.) is placed in a test tube and 1 c.c. of glacial acetic acid added and shaken; from 3 to 5 c.c. of ether are now added and shaken.

A portion of the ethereal extract is transferred to a small test tube and a small quantity of freshly powered guaiac gum is added and shaken; five drops of old turpentine (made by exposing chemically pure turpentine to the air for several weeks) are now added. In the presence of blood a blue to tokay color develops at the point of contact of turpentine and extract; or by shaking, the whole of the turpentine takes on the color.

The aloin test is made by adding old turpentine to a portion of the ethereal extract, and then 6 to 10 drops of an alcoholic solution of powdered aloin (a pinch of aloin in ½ to 1 c.c. of 70 per cent alcohol). The development of a bright red color within 5 minutes time at the line of contact of turpentine with the solution indicates the presence of blood. Beyond this time limit the aloin-extract mixture tends to change to red even though blood is not present.

Patients must be kept off a meat diet for three days previous to the collection of the feces; otherwise either test may react positively to the blood contained in the meat.

INTERPRETATION OF LABORATORY FINDINGS.

For purposes of diagnosis and especially prognosis, the interpretation of laboratory findings in pulmonary tuberculosis presents a difficult problem. Various tests have been brought forth with startling claims, only to be discarded entirely or to be relegated to the position of a link in the chain of evidence upon which the judgment of the clinician is based. Arneth's index, the albumin reaction, and the diazo have a similar history, illustrating the truth of the previous statement. Further, if we keep in mind the pathology and the general course of the disease, it is difficult to understand why we should expect any given test to supply us with all the information desired in forming a judgment. Such opinion may be formed from a knowledge of the extent, activity, and probable duration of the disease process.

The findings in blood, sputum, urine, and feces are but secondary to the disease process, and are of importance only as evidence of its extent and activity at the time of observation. Only in a very general sense can this information be of prognostic value. Nor may it be expected that this unsatisfactory condition will be remedied until more delicate serological and chemical methods have identified the primary principles conditioning infection and resistance to infection. Probably this information will come largely through increasing knowledge of the internal secretions, and cell ferments. Working under such limitations, we must not expect too much from the interpretation of our data. The making of diagnoses a little earlier and more confidently; the more certain determination of the nature of the complications which arise in the course of the disease; the collection of data which at best is only of limited value in establishing a prognosis; the accumulation of information, which, though of little or uncertain value in treating the individual patient, may in the aggregate throw much light upon the tuberculosis problem as a whole; these results are about all that may be expected.

Diagnostic and Prognostic Value of Individual Laboratory Findings.—The weight of individual findings varies somewhat with respect to whether or not bacilli have been found in the sputum; so, in the following discussion individual tests will be considered: first, as found in the prebacillary stage of the disease; and second, as found with bacilli in the sputum, after cavitation has taken place. For the most part, such findings are of presumptive evidence only, as they are not wholly specific for tuberculosis.

Sputum.—The nearest specific finding is a high lymphocyte content in the sputum. Fifty per cent or more of these cells indicate tuberculosis in probably over 90 per cent of cases, although theoretically a number of other lung conditions should give the same picture. As yet, however, nothing has been reported to minimize the importance of this finding as pointing to tuberculosis. The later development of bacilli in the sputum of some of these patients and the occurrence of the same picture in old healing lesions, is convincing.

The albumin reaction is less reliable as it is present wherever pus is found, unless it has been previously split by the action of ferments and bacteria in the lung. Such loss of albumin is found in conditions favoring retention of sputum, such as lung abscess and some cases of bronchiectasis. Chronic bronchitis gives little or no albumin in the sputum; acute bronchitis with high pus content gives a considerable reaction. Any albumin reaction then, persisting over a period of time longer than the course of acute infections, is due probably to tuberculosis. After cavitation has occurred, the presence of albumin is constant until healing has taken place.

Disappearance of Bacilli Under Treatment.—Of special importance to the patient and his friends is the permanent disappearance of bacilli from the sputum, during the course of treatment. The more extensive the involvement the more difficult it is to secure this result. Also, the better the technic employed in searching for bacilli, the less glittering will be the result. Rare bacilli, formerly overlooked by our cruder methods, are found today; so that the high percentage of patients positive on entrance but losing their bacilli under treatment, reported in past years, does not hold today. In 1909 a clinical report was published including all patients who had been in the sanatorium for at least 3 months. There were 277 patients who had bacilli on entrance, classified as I Stage, 12; II Stage, 26; and III Stage, 239. Of these, 11 of the I Stage, or 91 per cent; 16 of the II Stage, or 61 per cent; and 17 of the III Stage, or 7.1 per cent, lost their bacilli during treatment. The average course of treatment was 6 months for the I Stage; 8 months for the II Stage; and 10 months for the III Stage.

The sputum examinations at that time were made mostly from morning specimens, partly after direct smear and partly after fermentation of the specimen. A small proportion of the examinations were made from twenty-four hour specimens followed by fermentation. The collection of twenty-four hour specimens was started in July, 1907; although in patients with bacilli, morning specimens were considered sufficient. Beginning with July 17, 1911, twenty-four hour specimens were collected as a routine procedure, and the three-day specimen was adopted for all patients in whose sputum bacilli had previously been negative or rare. Since that time, in a total of 5160 sputa examined, 1150 have been three-day specimens.

Table XIII is a summary of all patients who were under treatment for at least 3 months, and in whose sputum bacilli were found at least once. The patients are arranged in two groups: those leaving prior to July, 1911; and those entering after July, 1911.

The number of patients in the I and II Stages above are too few in number to make valid comparisons; but in Stage III and in the total of all stages, a diminution in the percentage of patients losing their bacilli, during the course of treatment, is noticed when the better technic is used. The percentage is reduced from 8.4 per cent to 4.7 per cent for Stage III cases, and from 10.7 per cent to 8.5 per cent for all stages. The percentage of those negative on first examination but developing bacilli during the course of treatment is slightly less with the better technic, due probably to greater effort in making the first examination successful. While the percentage of patients who

TABLE XIII.

Showing Reduction of the Percentage of Patients Losing Their Bacilli Under Treatment, When Better Technics are Employed

Examinations before July 17th, 1911. Technic employed. Fresh morning specimens and 24 hr. specimens examined by direct smear and after fermentation. Preparations searched for 5 to 10 min. before reporting negative.					After July 17th, 1911 Technic employed. All specimens were 24 hr. or 3 day quantities, fermented and shaken in the mechanical shaker. Preparations searched for 15 min. before reporting negative.				
TOTALS	I STAGE	II STAGE	III STAGE	ALL STAGES	TOTALS	I STAGE	II STAGE	III STAGE	ALL STAGES
Patients	6	19	274	299	Patients	7	34	254	295
Examinations.....	43	91	2414	2558	Examinations.....	59	157	2522	2738
Average ex. per patient ..	7.2	4.6	8.9	8.6	Average ex. per patient...	8.4	4.6	10	9.3
Patients losing bacilli	3	6	23	32	Patients losing bacilli	4	9	12	25
Loss per cent..	50	31.6	8.4	10.7	Loss per cent .	57.1	26.5	4.7	8.5
Patients gaining bacilli....	0	3	10	13	Patients gaining bacilli. ..	2	0	9	11
Gain per cent..	0	15.9	3.7	4.3	Gain per cent	2.9	0	3.5	3.7

lose their bacilli has been considerably reduced, the percentage of favorable clinical results has been increased.

From these facts it is evident that the chances for a patient in Stage iii getting rid of his bacilli during the average period of treatment—9 months in the above series—is very small; and if still more sensitive technics were employed—pig inoculation or Petroff's cultural method—the chances would approximate zero. These cases must then be considered as possible carriers for a long period of time after treatment.

Another feature in the comparison of the two technics which does not appear in the table, is the higher percentage of patients who show bacilli intermittently by the better than by the poorer technic. There were eighty-four intermittent cases in a total of 295, or 29 per cent in the former; and 69 in a total of 299, or 23 per cent in the latter.

Blood.—Blood findings offer little aid to diagnosis in the prebacillary stage, except in comparatively rare instances where cavitation is preceded by severe constitutional symptoms; in such cases the almost specific character of the neutrophile stippling has enabled me to differentiate between tuberculosis and certain other non-tuberculous conditions. Rarely one meets a case of miliary tuberculosis presenting the same picture clinically; the presence of a leucocyte count below 5000 speaks for the miliary condition with a grave prognosis; a high leucocyte count—10,000 or above, depending on the severity of symptoms, rather indicates approaching cavitation, without prognostic significance. Arneth's variation in the neutrophiles is entirely nonspecific, occurring in practically all infections with constitutional symptoms. A high lymphocyte count was formerly looked upon as of diagnostic value, but so many other conditions produce the same picture that little may be expected of it; further, the discovery in later years that the average lymphocyte percentage in normal persons is much higher than was originally claimed by Ehrlich has also discounted the value of the lymphocyte percentage. After cavitation has taken place, however, there is a close parallel between the number of lymphocytes in the blood and the general welfare of the patient. The higher the count, the better on the average is the condition of the patient, and vice versa. For prognostic purposes a count of 500 or below indicates an almost certain fatal issue.

Urine.—The urine findings before bacilli appear in the sputum are of little value. The occurrence of a diazo and urochromogen are rare, and when they are present they are associated with the severe constitutional symptoms accompanying extensive softening or a miliary condition. These two conditions might at times be confused with typhoid fever accompanied by pulmonary and pleuritic symptoms. As the diazo is likely to occur in all these conditions, it is of no value in differentiating, but it is of some value in excluding pneumonia in which it occurs less commonly. The diazo is not frequent in tuberculous meningitis unless the condition is terminal to massive pulmonary involvement, so that it should be of some weight in differentiating tuberculous meningitis from typhoid fever, miliary tuberculosis and massive pulmonary involvement, when mental confusion is present.

The diazo is of limited prognostic value. An occasional occurrence indicates nothing more than that the disease is quite active at the time of examination. Continued occurrence of the reaction gives a grave prognosis. Two patients in whom I found a positive test at $\frac{1}{5}$ dilution of the urine, during cavitation, never gave a diazo thereafter, and obtained complete arrestment. Both are alive and well, one after 6 years, the other after 2 years; on the other hand, a diazo of such intensity persisting for one month is almost certainly fatal within a few months' time.

Indican findings, considered alone, are of uncertain value in the individual case, although an increased indican elimination has long been known to accompany tuberculosis. There is considerable variation in the amount of indican eliminated in twenty-four hours, so that little information is likely to be obtained from a single examination. Some years ago I determined the amount of indican in three successive daily twenty-four hour urines from 45 different patients. Comparing the results for the three days it was found that in 6 patients the highest reading was less than 25 per cent greater than the lowest; in 3, $33\frac{1}{3}$ per cent; in 11, 50 per cent; in 16, 100 per cent, and in 9 more than 100 per cent. In interpreting indican findings, one must know whether or not a cathartic was given before or during the time the specimen was being saved; and also as to the result of the cathartic. Should one find a persistent increase of indican in a patient with bowel symptoms, tuberculous enteritis should be thought of; these patients almost invariably have indican values above 30 (15 being normal; see Indican determination, p. 574) even though on a restricted proteid diet.

The chief value of indican determinations has been as an aid in the regulation of the diet of the institution as a whole, as well as the diet of individual patients. In 1908, a study was made of indican reports made up to that time: the first examination on entrance was compared with the average of all succeeding examinations from each patient. It was found that the average increase of indican in 69 patients was from 15 to 26, while the average decrease in 32 patients was from 26 to 14. The patients were then classified with reference to their general condition and habits of eating. It was found that those whose indican increased markedly on our diet were almost invariably the ambulatory patients who were trying to carry out faithfully the erroneous teaching of "stuffing." The practice had been discarded several years before in the institution, but it could hardly be expected to die quickly after having been advanced and maintained with such insistence by the profession. In many cases patients were told by the physician referring them that what they needed was good, nutritious food in excess of what their appetites called for. It was not an easy task to break up a habit which had such a strong reason for its existence. However, the high indicanuria in at least two-thirds of the patients was so clearly dependent upon the excess of proteid food ingested, that effort was made to break up the habit. After a course of general lectures, with a careful watch over individual cases, the indican determinations were compared as above from the next 76 patients entering, with the result that only 23 patients showed an increase of from 22.1 to 27.4, while 53 showed a decrease of 27.3 to 14.0. These averages are made occasionally, whenever there is reason to believe that the patients have forgotten the previous lesson.

The following chart shows a curve made from the averages of all indican values found for each month for seven years. The technic has been uniform throughout. From 60 to 80 examinations are averaged each month, so that the chart represents the findings in about 5500 twenty-four hour urines. (See Fig. 102.)

On an average the curves show a lower indican elimination since 1912. It was before that time that we experienced most of our trouble from overeating. Aside from this tendency, there are certain other causes tending to influence the indican curve. In April, 1912, a sharp rise in the curve followed a period of warm days entirely out of season. The remaining months of the year were cool. Aftér a cool spring and summer in 1916, a period of two weeks of warm weather were experienced in

August, with a rise in the curve. This rise in the indican curve, with the usual train of minor symptoms, is the same condition met in the eastern states with the approach of the warmer spring months. With us it is likely to occur at any time, due to our lack of well defined seasons. Just what atmospheric conditions are responsible for this rise in the curve is not clear. It is not the temperature alone; for low indican values are often found in hot weather. It may be due largely to the effect which certain atmospheric conditions have in increasing bacterial activity, rendering food less digestible, disturbing the digestion of the patients and increasing intestinal intoxication.

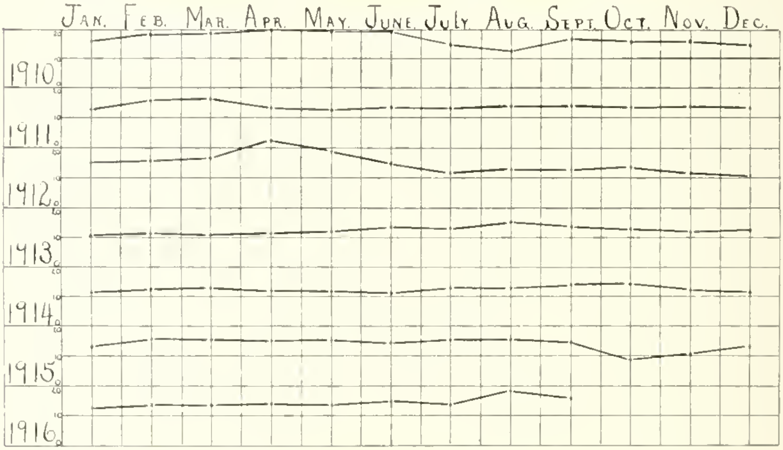

Fig. 102.—Institutional indican curve determined from the average of all indican findings for the month.

Whatever the exact explanation, the curve is of much value in guiding our patients through these periods of disturbance. They are advised to diminish the proteid content of their diet.

Tubercle bacilli in urine, whether from the kidney or elsewhere in the urinary tract, give a grave prognosis; although cases have been reported as having healed. Bacilli have been reported as having been found in the urine in miliary tuberculosis, without involvement of the urinary tract; but as such cases are rare, confusion is not likely to result. The presence of pus with bacilli in the urine justifies the opinion that tuberculosis exists somewhere in the tract.

Feces.—The diagnosis of tuberculosis of the bowel from the presence of tubercle bacilli alone in the feces should not be made; even though they are continuously present. In case bowel symptoms are present, the condition should be thought of; but the diagnosis can by no means be established for a certainty. The continuous presence of occult blood in a patient with advanced pulmonary tuberculosis, provided ulcer of stomach or duodenum can be excluded, rather points strongly to tuberculous enteritis, if clinical symptoms are also present. This complication is rarely recognized until the disease is far advanced.

Correlation of Laboratory Findings.—In the proper correlation of findings from all sources, is to be found the chief value of laboratory work; in such a method negative findings by individual tests are often as important as are the positive findings obtained by other tests; the varying pathological condition of the patient causes a corresponding change in the laboratory findings; so that negative findings often enable the clinician to exclude certain disease processes from consideration in diagnosis, while positive findings give him an idea of the nature and extent of the condition itself.

When the constitutional symptoms are slight, the laboratory findings vary little from normal; but when they are pronounced, whether bacilli appear in the sputum or not, we must differentiate between tuberculosis and other respiratory infections. Even though we are positive that the clinical picture is one of uncomplicated tuberculosis, the assembling of the laboratory facts gives a more accurate idea of the condition of the patient. In the sanatorium it is rarely necessary to differentiate tuberculosis from the acute respiratory infections on entrance; as 95 per cent of all entrants have been under the care of their physicians for sufficient time to exclude almost entirely the latter diseases. The following case was an exception to this rule, as the patient was delirious and his family were unable to give a satisfactory history; they rather attributed his mental state to business worries.

On entrance the patient was delirious; temperature 104.4, pulse 124. Physical examination in recumbent position was unsatisfactory, giving weak breath sounds over right apex.

Sputum: small amount; purulent in character; no bacilli present. Urine: 24 hr. 1000 c.c.; albumin, large trace; diazo at 1/6 dilution. Blood: white cells 4780; neutrophiles 74 per cent; lymphocytes 24 per cent; neutrophile stippling, moderate. It was observed at the time that the stippling was unlike what I had found in severe toxemias in uncomplicated tuberculosis and a note was made of the differences. The granules were neither as distinct nor as black in color, as is invariably found in the latter. Bacilli were not present in the feces.

The clinical picture presents these possibilities: tuberculous meningitis; miliary tuberculosis; massive pulmonary tuberculosis; some acute respiratory infection, as pneumonia or influenza; typhoid fever with pulmonary complication; and finally, any of the infectious diseases with pulmonary complications in a person who was previously mentally deranged. The marked diazo reaction with leucopenia almost certainly limits the possibilities to miliary tuberculosis, massive pulmonary tuberculosis, and typhoid fever; but the physical examination, though unsatisfactory, almost certainly excluded pulmonary tuberculosis of such degree as would be necessary to produce such toxemia. The differentiation of miliary tuberculosis and typhoid fever was made in favor of the latter, on the character of the neutrophile stippling, as not due to the toxemia of tuberculosis. Two days later rose spots appeared on the abdomen. Repetition of the laboratory tests 5 days later gave a diazo at only 1/3 dilution; white cells 4050, bacilli negative. The blood culture for typhoid bacilli was not made. Sputum and urine were examined later 3 different times, with the result that bacilli were never found and the diazo was absent at the end of three weeks. The subsequent history showed that we were dealing with typhoid fever. The temperature remained between 104° and 105° for 13 days, coming gradually to normal on the 21st day, and remaining normal thereafter for four weeks, except for an occasional rise to 100°. In this case the most specific finding was the character of the neutrophile stippling, which is suggested for the use of those workers who have more opportunity for differentiating these cases than I have had.

In the following case, I believe the laboratory findings were important not only in confirming the physical findings, but in indicating a more accurate prognosis than would be ventured from the former. There was no question of the diagnosis of tuberculosis, although bacilli had never been found. The patient gave a history of cough at times with or without sputum. Six weeks before entering the institution he had a "cold" lasting for three weeks, with a maximum temperature of 102° to 104°, and subnormal in the morning. Physical examination showed extensive infiltration in both lungs. The temperature on entrance was 99°; pulse 90. Sputum, 24 hour specimen, gave no bacilli. Urine, no diazo. Blood, white cells, 3850. Because of the leucopenia, the white cell count was repeated the next day, giving 3900. Three days later it was 5700; lymphocytes 910; Arneth's index 97. Three days later, the count was 3500; a diazo at ½ dilution was found in the urine; and a 3 day sputum was negative for bacilli. At this point the temperature was 102° without morning remission, continuing in this manner until death 10 weeks later. Laboratory examinations were repeated 8 days after the last examinations. The white cell count was 4050; diazo 1/7; tubercle bacilli 25 per field. The white cell count repeated 10 days later was 4700, and 8 days after that, 6050. Seven weeks after entrance the urine gave a diazo of 1/12; the sputum 75 bacilli per field.

The main point of interest in this case is the persistent leucopenia, associated with the increasing intensity of the diazo, which justified the opinion of an early fatal issue, which was not indicated by the history and physical findings. The second complete physical examination, made one month after entrance, showed that areas of infiltration had increased markedly. This was a case of miliary tuberculosis of the lung.

Since developing the special technics for the determination of the sediment volume and the length index of tubercle bacilli, I have found the findings very valuable in explaining the nature of the lesser changes taking place in the patient's condition from day to day. Those who are engaged in following the course of the disease in tuberculous individuals, know of the vast number of little complaints of such patients, such as ½ to 1 degree rise in temperature without evident cause; the tendency to tire easier on certain days as compared with others; loss of appetite, etc. These complaints come from those who are comparatively well, as well as from those who have considerable involvement with constitutional symptoms.

There has been some speculation concerning the meaning to be attached to the relative length of bacilli found in sputum; the two important opinions being: first, that the short uniformly stained forms are of the bovine type, while the long and beaded forms are of the human type; and second, that the short and long forms are the younger and older bacilli respectively. Although I have kept routine records of the estimated relative length of bacilli for eight years, I came to the conclusion that it was practically impossible to form an idea of the relative length of bacilli, without some scheme for measurement. Since perfecting such a device, I have been able to classify bacilli to within a narrow margin of error. I find that many of the complaints referred to above are associated with the softening of minute areas in the lung, as is shown by the increase of the short bacilli. We have in this method a means of detecting these slight changes, which neither the stethoscope nor other means, of which I know, can determine. Such information is of great value in individual cases, such as those showing very irregular temperature curve, and in which there is doubt as to whether the temperature is referable to the lung involvement, to some tuberculous complication, or to some non-tuberculous process.

To rely on the patient's statement that he is coughing and raising more today than he did yesterday or last week often leads the clinician to wrong conclusions. Changes in atmospheric conditions may produce those symptoms, and may even produce a slight rise in temperature. Further, worry, fright, anger or other emotion may either directly or indirectly affect the temperature curve, producing a train of symptoms besides the ones mentioned. The differential diagnosis in these cases has repeatedly been made to the satisfaction of the clinicians and the patient himself by means of the sputum findings as illustrated in the charts. (See Figs. 103 and 104.)

In addition to the temperature curve the charts present 5 curves made from quantitative observations extending over several months. The temperature curve was made from the averages of the highest temperature for the day, of three successive days. This method minimizes the influence which various outside factors would have on the curve. For instance, indigestion and the various emotions, may cause a rise for a single day of one or two degrees, but when averaged with two days of usual temperature, it would not appear of much importance on the chart.

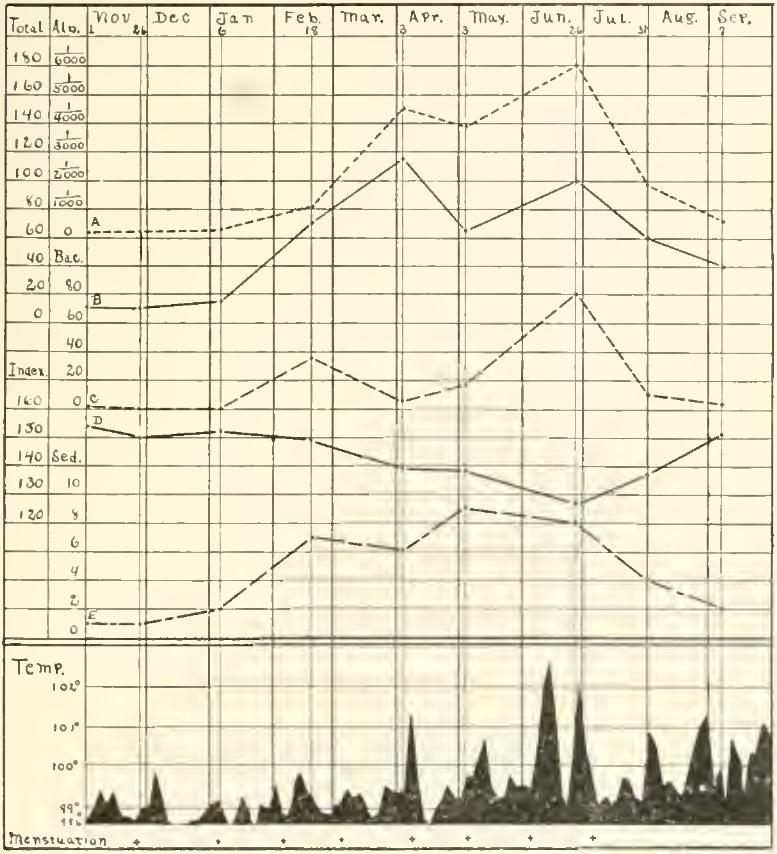

Fig. 103.—Chart showing correlation of sputum findings and their relation to temperature curve. *A*, Albumin curve; *B*, total 24 hour sputum; *C*, number of bacilli; *D*, length index; *E*, sediment volume.

Fig. 103 presents the findings in a female patient whose lung involvement has extended under treatment. It will be noticed that in general the albumin, total quantity of sputum, relative number of bacilli, and sediment curves are in agreement with the temperature curve. The length index curve bears an inverse relation to the other curves; that is the greater the activity the shorter the bacilli, and the lower the index. This relationship is shown in Fig. 104 and in some 40 other charts, which I have made from other patients, establishing beyond all doubt the meaning of short bacilli, and confirming the opinion of others who have expressed this view. This view does not necessarily weigh against the other opinion that short bacilli represent one type and long bacilli another type. The greatest fluctuation in

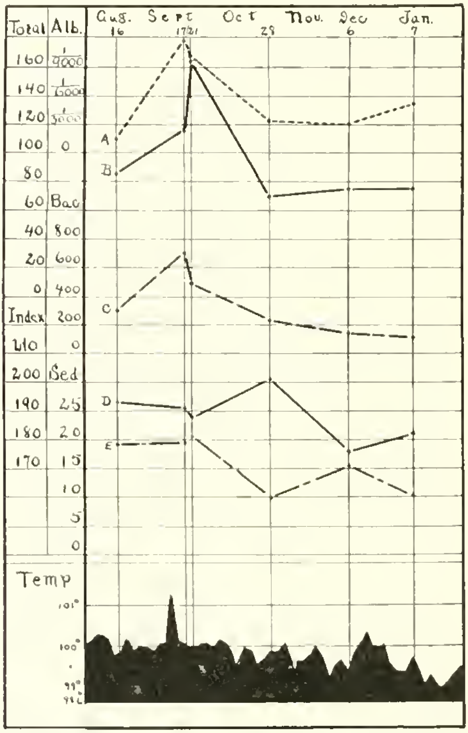

Fig. 104.—Chart showing correlation of sputum findings and their relation to temperature curve. *A*, Albumin curve; *B*, total 24 hour sputum; *C*, number of bacilli; *D*, length index; *E*, sediment volume.

index which I have met in any single patient was from 264 on entrance to 204 after cavitation two months later. Another patient on entrance gave an index of 177, and after two months of unusual progress, an index of 218 was found. Some of these patients have been under observation for 10 to 15 months, and while the index varies considerably, it does so within fairly narrow limits—50 to 40 points in index—which is not great considering the possible range from 127 to 264 as found in my work. This rather points strongly to a number of different types of organisms.

I have not been able to find any special significance in the albumin curve, and the total 24 hour specimen varies so greatly that its curve is of little value in studying these finer points in diagnosis; so that in practice, the sediment volume, length index, and relative number of bacilli are given most weight.

CHAPTER XXI.

THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF TUBERCULOSIS, PARTICULARLY PULMONARY TUBERCULOSIS.

The diagnosis of tuberculosis is so intimately associated with its differentiation from other conditions that it seems logical and best to discuss these subjects together.

The Importance of Diagnosis in Hidden Tuberculosis.—While our chief duty as clinicians, as yet, so far as tuberculosis is concerned, is to diagnose the disease as it affects the lungs, yet we must not lose sight of the fact that it may affect any tissue or organ of the body; and that each lesion may produce the same group of toxic symptoms together with a group which will be characteristic of the particular tissue or organ affected. It is especially necessary to bear this in mind if we would understand the indefinite group of toxic symptoms which accompany a partially quiescent focus in the peribronchial or tracheal glands where the main complaint of the patient is that which might be caused by any slight toxemia. In such cases we have the picture of loss of tone and lack of nervous and physical force without any apparent cause. In malarial districts malaria is usually diagnosed, while, in other places neurasthenia, overwork, or a general rundown condition is supposed to be the cause.

As clinicians we are apt to forget the possibility of such symptoms being caused by hidden foci; and, failing to find tuberculosis in the lung, declare that it does not exist. We should never lose sight of the fact that, with clinical symptoms belonging to the toxic group suspicious of tuberculosis, and an inability, by most painstaking examination, to find either a tuberculosis or other cause for the same, we have not ruled out the possibility of a hidden tuberculous focus. The pulmonary tissue is not the only part attacked by this infection; in fact, the peribronchial and peritracheal glands are nearly always infected prior to the pulmonary involvement and symptoms can arise from the escape of toxins from these glandular foci, even although the glands may not be enlarged sufficiently to cause symptoms other than those of a toxic nature, and even though they cannot be outlined on physical examination. It is equally important to remember that, given a lesion in some organ other than the lung which is suspected of being tuberculous, with undeniable evidence of tuberculosis being present in the body of the patient, such as a positive tuberculin reaction; or, a positive sputum finding, this positive evidence does not prove the tuberculous etiology of the process in question.

Difficulties of Diagnosis.—The difficulties which attend the diagnosis of tuberculosis come from many sides. The fact that tuberculosis has been a fatal disease for so many centuries still militates against its detection. Although tuberculosis *if diagnosed early and treated intelligently* has been removed from the list of fatal diseases, yet this is only accepted in a half-hearted manner. It is not fully believed by either the laymen or the profession. This lack of faith in its curability adds to its deadliness because it keeps up such a fear of the disease that the patient and his friends fight against knowing the truth, and medical men shrink from finding it out, or, if they know it, from telling the patient until the early curable disease has become an advanced hopeless one. This can only be remedied by medical men accepting what intelligent treatment can do for truly early tuberculosis and appreciating the great danger of delay; and then proclaiming it far and wide until the truth is fully known.

It requires Herculean effort to work against the tide of universal public opinion; yet, this is the task imposed upon those who are striving for early diagnosis and cure of tuberculosis.

The insidiousness of tuberculosis is also a factor which makes the real diagnosis difficult. If symptoms came on at once after infection took place, it would be comparatively an easy matter to make an early diagnosis, but such is not the case. Infection in tuberculosis takes place weeks, months, or years before clinical symptoms are recognized, the lesion having gone through a succession of changes from quiescence to activity and extension.

This goes on until the process is sufficiently extensive or until the activity is sufficiently great to produce symptoms which are unmistakable. It is not the frank, unmistakable symptoms which we should look for. These are evidence of advanced disease. We should always, bearing in mind the frequency and insidiousness of this disease, be on the alert and learn to recognize the earliest symptoms which occur.

If only there was some group of symptoms which is pathognomonic or some specific tests which are infallible in their diagnosis of early tuberculosis the problem could be minimized. But there is not; so we are often obliged to make a diagnosis by exclusion rather than by positive data.

The symptoms of early tuberculosis differ according to the nature and location of the lesion. Toxins produce many of them; but the lesion must be of some extent before these are present in sufficient amount to be evident; and in some locations the symptoms caused by the inflammatory process are more marked. In such locations as the meninges, bones, joints and pleura, symptoms dependent upon toxemia are comparatively unimportant at first; so are they often in glandular tuberculosis, and even in the early pulmonary form. On the other hand, if the disease is located in the tissues where it can run a chronic course, sooner or later the toxic group of symptoms becomes important. In marked glandular tuberculosis and in pulmonary tuberculosis, beginning with the so-called early lesions, which are in reality lesions which are more or less extensive, toxemia is usually accountable for a definite group of symptoms which must be considered in the diagnosis; but, unfortunately, these are not constant.

In genuinely early lesions there is no definite known blood picture; there are no secretions to analyze; and the specific reaction of the body cells and tissues to the products made from the tubercle bacillus (tuberculin) is usually, though I believe wrongly, considered as being of no value except in the first few years of life. The x-ray can give evidence, which, if rightly interpreted, may be of value in early infections located in some tissues, particularly the joints and bones, and at times plates made by the best apparatus with expert operators will greatly aid in the diagnosis of lesions in early pulmonary invasions.

The Importance of the Tuberculin Tests in Diagnosis.—The function of the tuberculin tests in diagnosis is not understood because there are so many things about the specific reaction between tuberculin and the sensitized body cells that we do not know. However, it is true that we are arriving at some definite opinions. Our judgment of them, now that we know the frequency of tuberculosis in early years and understand its tendency to go through cycles of activity and quiescence over a long period of time until it either heals or produces frank clinical tuberculosis, should help us in at least having respect for a reaction when found, even if it does not afford us the help that we desire at the time.

What clinician can say, by the most careful minute examination that a hidden focus of tuberculosis is not present in some portion of the patient's anatomy? Yet it is an every day occurrence for physicians to make such statements and put up such opinions against positive tuberculin reactions. They do not presume to do so in syphilis. If a positive Wassermann is found, the clinician accepts the diagnosis whether he is able to find a trace of trouble clinically or not; yet, he is no surer in this case than he is in the case of the tuberculous patient. The answer to this is that the tuberculin reaction is not absolute, but the Wassermann is. How has this been proved? Is not a hidden active tuberculous focus as dangerous as a hidden active syphilitic focus, and is not a hidden active tuberculous focus as dangerous as a detected one? Have we any grounds for belittling the danger of such a focus? The fact that all such foci do not become serious clinical entities and do not go on to the death of the individual, is not sufficient, and would not be accepted in any other field of medicine. The fact that tuberculosis infects probably two-thirds of all who reach adolescence; and that it produces toxins which impair the health of a considerable per cent of those infected; and that it is the cause of death in about one-tenth of the world's population; and that it causes a morbidity four times as great as its mortality, should be sufficient to emphasize the importance of all such foci.

If only we could determine in some manner how long specific cell stimulation or sensitization remains after inoculation, or particularly after healing of a tuberculous focus, then we could arrive at some definite opinion as to the value of tuberculin in diagnosis. Until such time, however, we are obliged to make our medical imagination, which when interpreted, means our ability to theorize and make clinical application of our theories, take the place of definite scientific facts. From many years of observation in the employment of the tuberculin tests, particularly in cases of pulmonary tuberculosis, the writer has developed a firm belief in the value of these tests. If only we bear in mind that the reaction is an expression of specific cellular defense (cell sensitization) against the bacillus and its specific products, we will understand the interpretation of the phenomena better. The cutaneous and intradermal tests are the ones that I prefer to use and the ones which I think are easiest of interpretation.

It is thoroughly established, according to the observations made in the study of immunity, that an individual fights infection by creating a specific cell defense. This specific defense is, to a certain extent, commensurate with the severity of the inoculation and the reactive capacity of the body cells, which latter is bound up quite closely, as far as we are able to determine, with the degree of health of the individual. All else being equal, the strong robust patient reacts best and the reaction is greatest when the infection is not too severe.

If these observations are correct, barring the very severe inoculations, we should expect most active defense when there is the greatest need, that is, when fighting an active infection; and the greater defense, the more resistant the individual. We would also expect this defense to show as an increased sensitization (an increased specific defensive power) of the cells toward the bacillus or its products. Tuberculin, containing the specific bacillary products, should, then, when brought in contact with the body cells, provoke a reaction commensurate with the degree of specific sensitization (specific defensive powers) present, being greater when the patient's reacting power is good and when fighting an active lesion, and less marked when the lesion is quiescent, and, theoretically, at least, finally disappearing after healing has occurred.

This has been my experience in practice. By interpreting frank prompt reactions as positive evidence of an active tuberculous focus somewhere in the body, one will not go far wrong. Such interpretation will not be farther from the truth than our usually accepted methods in other lines.

This leaves a very important class of cases in doubt, however; for there are individuals whose body cells fail to react properly in building up specific defense. In these, accurate diagnosis and proper therapeutic measures are even more important than in those whose defense is good, but the tuberculin test may prove valueless. This will be in a smaller proportion of cases than is generally believed. The cachectic, those severely ill, those with atrophied skin and those suffering from acute illness other than tuberculosis, particularly some of the acute infectious diseases, will remain in doubt. But, knowing this, why not use the test for the definite knowledge it gives. Every practitioner of medicine has certain limitations in diagnostic and therapeutic ability, but he does not refuse to examine and treat all diseases because of it. He secures other help to cover his weakness.

Let it be understood that the power of bacillary products (tuberculin) to seek out and stimulate specifically sensitized cells may be far more accurate than our power to locate and determine the activity or quiescence of tuberculous foci; and, when such evidence is present, even though we have not been able to locate the lesion, let us accept it as a tangible proof of our limitations rather than an instance of nature's specific reaction gone wrong.

Variability of Tuberculins.—At this point permit me to call attention to an important factor which is probably militating against exactness in the tuberculin tests. We know that different skins react differently; that the degree of reaction depends upon the reactive powers of the patient; that it is influenced by certain infections; and that it is further influenced by the method of making the test; but I desire to call attention to another source of difficulty in interpretation which lies in the tuberculin itself. Tuberculin made by different manufacturers differs greatly in its strength and in the percentage of component parts of the tubercle bacillus which it contains. During recent years I have made a careful study of the character of the reaction following the von Pirquet test. In this I have used Koch's Old Tuberculin, full strength. I have found that the preparations made by some manufacturers would give a reaction twice or three times as strong as the tuberculin made by others. It is very desirable to have some method of standardizing these reactions. If we have a tuberculin which contains comparatively few of the principles of the tubercle bacillus, which are active in producing a reaction, we can readily see that the maximum reaction from this preparation would be decidedly small, as compared with one which was rich in these active principles. After observing this fact I made the comparison of several different preparations and found that the tuberculin made by one manufacturer gave a maximum reaction of 3/4 cm. in diameter, in the same patient in which a preparation made by another manufacturer gave a reaction of 1½ to 2 cm. From this, it can be seen that the reaction of ½ cm. would be considered a moderately severe reaction, when interpreted in the terms of the second preparation.

The importance of this is evident because a prompt maximum reaction accompanies activity in a tuberculous process in an individual with good reactive powers, while a slight reaction coming on slowly, is rarely found in one fighting an active infection.

The diagnosis of early active pulmonary tuberculosis is not an easy matter unless the infection possesses considerable virulence and results in the formation of many toxins, or the lesion is fairly extensive. We must disabuse our minds of the thought that we are diagnosing the disease as soon as it affects the pulmonary tissue. Occasionally it is weeks, but more often months and years after the implantation of the bacillus and the production of the first fibroid area that activity again shows itself, and an extension of the disease takes place with a production of the symptoms which we have learned to recognize as belonging to early pulmonary tuberculosis.

The examination of the lung, however, can be approached in so many ways that when our data is all correlated, we are in a position to give a fairly accurate opinion. By the time the information obtained from clinical history, physical examination, sputum examination, tuberculin tests, and the x-ray are correlated, the per cent of error will be no greater than in any other field of diagnosis, made by an examiner of equal skill.

Importance of Clinical History in Diagnosis.—The importance of obtaining an accurate clinical history seems to be generally underestimated, at least in practice. While it is true that some of the symptoms of which the patient complains are of little differential diagnostic worth and could be produced by other pathological conditions, yet there are others that have greater significance. If the history is carefully and fully taken, there will usually be enough symptoms elicited to point definitely to a pulmonary tuberculosis and make such a diagnosis a probability.

In the grouping of symptoms according to their etiology[1] I have endeavored to show the relative importance of the different groups of symptoms of early tuberculosis and their diagnostic value. Those due to toxemia such as malaise, feeling of being run down, lack of endurance, nervous instability, indigestion, and loss of weight, rapid heart action, night sweats and temperature do not point to anything definite. They could be caused by a pulmonary or a glandular tuberculosis or a lesion in most any tissue of the body, or to a non-tuberculous toxemia of light degree. Consequently, they alone are of little diagnostic aid. But, when the lung is involved, they rarely occur alone. One or more of those of reflex origin, such as hoarseness, tickling in the larynx, cough, indigestion with loss of weight, chest pains, particularly aching of the shoulders and over the apices and upper portion of the lung, increased pulse rate and flushing of the face, are almost sure to be present if the focus is active; so, also, one or more of those due to the tuberculous process *per se*, such as frequent and protracted colds, spitting of blood, pleurisy, sputum, and rise of temperature.

Hoarseness or tickling in the throat, when due to a tuberculous irritation, with or without cough or expectoration, is nearly always accompanied by some of the toxic symptoms above mentioned, and this combination should point to the lung. Local lesions in the larynx of a simple nature which cause persistent hoarseness or inclination to cough are not apt to be accompanied by toxic symptoms. When sufficient irritation is present in the lung to produce reflex hoarseness and cough, either the diaphragm reflex or the spasm of the superficial muscles, or both, are practically certain to be present; and, if so, add greatly to our suspicion of an inflammation in the lung. The more experience I have in the study and employment of the reflex motor phenomena, as they are found in active pulmonary tuberculosis, the more I depend upon them when in doubt.

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[1] The Altered Condition of the Neck and Chest Muscles and Subcutaneous Tissue Overlying Them as Important Aids in the Early Diagnosis of Tuberculosis, Northwest Medicine, 1915, vol. vii. No. 6, June; and A Classification of the Symptoms of Early Pulmonary Tuberculosis Based on Their Etiology, St. Paul Medical Journal, 1915, vol. xvii, No. 1.

A failure to appreciate the nature and cause of the symptoms and signs which accompany tuberculosis has caused great confusion in the examiner's mind and made the diagnosis of this disease extremely difficult. Now, however, that we have classified them etiologically, we can better understand the different manifestations of the disease and form a more accurate conception of the underlying pathology as expressed in the symptomatology.

In all organs which are supplied by the sympathetic division of the vegetative system with inhibitory fibers, and by the parasympathetic with activating fibers, there is very likely to be during periods of toxemia, a general depression of function, because the sympathetics, being centrally stimulated, produce such an increased tonus that they are apt to overcome the parasympathetic tonus and destroy the normal physiological balance.

The importance of understanding the nervous system in both its relationship to the lung and to the toxins engendered by the tuberculous process cannot be overestimated. Two of the groups of symptoms which I have described are expressions of nerve stimulation. The symptoms in one of these groups,—those of toxic origin—are due to central stimulation; while the other,—those of reflex origin,—are due to peripheral irritation. This difference, naturally, stamps these groups with a distinct individuality. In this connection, however, we must not forget the peripheral effects produced in the tissues supplied by the sympathetics resulting from adrenin.

Symptoms belonging to Group I (toxemia) are expressions of central nerve, and endocrine stimulation plus a general discharge through the sympathetic nervous system and manifest themselves in many organs and tissues at the same time. They are also present under several conditions.

1. When bacilli in the tuberculous focus are multiplying and toxins are diffusing into adjacent tissues; likewise when necrosis and caseation are taking place.

2. When, after the acute inflammation has disappeared, through wrong methods of living, particularly overexertion, autoinoculation produces sufficient toxemia to continue the irritation of the sympathetic nervous system.

3. When, through prolonged action of the toxins the normal sympathetic balance has been more or less permanently disturbed and a condition of neurasthenia has developed.

4. When, through fear, discouragement and disappointment, as a result of the disease or for some other cause, general sympathetic stimulation takes place.

While toxic symptoms are due to central stimulation of the sympathetics, and are prolonged by peripheral stimulation by adrenin, reflex symptoms are produced by stimulation of the peripheral endings of both the parasympathetic and the sympathetic divisions of the vegetative system in the inflamed area in the lung. These symptoms are also widespread, affecting many tissues and parts; but they express themselves with a wide degree of variability. At one time or in one organ the symptoms may be the expression of a parasympathetic reflex, at another time and in another tissue they may be that of a sympathetic reflex. When the action of both divisions affects the same organ, as it does in the heart, stomach and intestines, parasympathetic tonus may predominate at one time and sympathetic at another; and at still other times, the stimulation through the two systems may balance each other and maintain a normal equilibrium.

When acute toxemia is present the central stimulation of the toxins on the sympathetics, added to the peripheral irritation of the sympathetic nerve endings by the increased adrenin in the blood which acts at the myoneural junction, overbalances the vagus tonus in many instances, with a resultant general depression of function in the viscera. We now and then see notable exceptions to this rule, however, in that the pulse rate will remain lower than is common in other fevers, of equal degree, during acute cavity formation in the lung. Hyperacidity will now and then also increase in severity during this stage of increased toxemia. The explanation for this is that the reflex irritation of the parasympathetics is sufficient to preserve a strong condition of tonus which if not sufficient to overcome the effect of the sympathetics, at least is sufficient to greatly modify it. The absence of symptoms of the toxic group does not mean that active tuberculosis is not present.

The symptoms due to reflex cause are variable; at one time a sympathetic tonus may be most prominent; at another a parasympathetic reflex may predominate in the same organ. When the conditions which are accountable for the toxic symptoms disappear, what symptoms remain depend on whether parasympathetic or sympathetic tonus predominates. We now find a rapid pulse due to increased sympathetic tonus; now one slower than normal due to increased parasympathetic tonus; now depressed secretion and motility in the stomach and intestines as a result of increased sympathetic tonus; and, again, an increase in both functions due to increased parasympathetic tonus. This antagonism is present as long as the peripheral nerve endings in these two systems are irritated and the central cells are stimulated.

We should probably not go far wrong if we assumed that permanent injury follows this chronic stimulation; and, that some of the degenerative changes which manifest themselves in the internal viscera of the patients suffering from chronic tuberculosis, are due to this reflex irritation. We have analogous grounds for suspecting degenerative changes in the internal viscera, in the trophic disturbances which manifest themselves in the skin, subcutaneous tissue, and muscles, as a result of reflex sympathetic irritation, as already described.

The symptoms due to the tuberculous process *per se* are the most trustworthy symptoms in early pulmonary tuberculosis, and one, or more, is usually present comparatively early in the disease, although they rarely show as early as those of toxic and reflex origin. Frequent and protracted colds are commonly present, but rarely occur except when a fairly extensive lesion is present; but, when present, should always make us think of pulmonary tuberculosis. These are nearly always accompanied by some rise in temperature and some of the symptoms of the other two groups. Spitting of blood makes the diagnosis almost positive unless some other cause can be found; so does pleurisy. Pleurisy and spitting of blood may both come on suddenly with few or no symptoms belonging to the other groups, although, of the two, pleurisy is more apt to be so accompanied. If, instead of calling pleurisy by its simple name we would call it by its true name, *active tuberculosis of the pleura*, we would have far more respect for it and often give our patients the advantage of having a tuberculous involvement diagnosed when it first announces its presence. Sputum containing bacilli, while rarely present, may be found, occasionally, very early to the great surprise of the examiner. This follows sufficiently often to make it imperative on the examiner to collect all sputum raised by the patient, regardless of his opinion as to where it comes from, and subject it to one of the more accurate tests for bacilli. A three days' quantity should be taken in all such cases and treated as described fully in Chapter XX.

Temperature as a diagnostic sign in tuberculosis has lost some of its long time suspected value. Formerly, clinicians were willing to make a diagnosis of tuberculosis on a slight persistent rise of temperature which could not be assigned to any other cause, even if no other sign of the disease could be found. Now, that we know there are many lesions of localized infection such as those produced by microorganisms of the streptococcus type, and have seen many postinfluenzal lung infections which may cause persistent slight elevation of temperature, we are coming to realize that differentiation is necessary. Such rises in temperature must be looked upon as being a part of the general syndrome of toxemia, and as such, may be caused by infections of the tonsils, teeth, sinuses, prostate, fallopian tubes, or any other organ.

In instances of hidden tuberculous foci and hidden infections of other types, differentiation may be very difficult for we may have the same group of toxic symptoms and the same type of temperature curve. In such cases the value of the tuberculin test must not be forgotten. A positive well marked cutaneous reaction indicates such a sensitized condition of the cells as accompanies an active tuberculous focus; or a reaction to the subcutaneous injection indicates the same. Of course, infections of both types might be present and this possibility must not be overlooked.

From our discussion it is now plain that the only group of symptoms which, unaided, permits of the making of a definite diagnosis are those belonging to the group caused by the tuberculous process *per se*. But, fortunately, if the history is taken with sufficient care, any one important symptom or any group of symptoms will most likely be fortified by others. A fact which is often lost sight of is that it is just as important to question the patient further regarding points in his clinical history in case of doubt, as it is to reexamine the chest, or sputum. Patients are rarely able to give a complete, accurate, clinical history upon the first interrogation.

What Value Has Physical Examination in Diagnosis?—The physical examination of the chest is the procedure upon which the decision as to the presence or absence of pulmonary tuberculosis finally rests. When one recalls the few changes which small early lesions cause in the pulmonary tissue, and the difficulties which beset the usual methods of examination, he is forced to believe that reliance on physical findings by men with insufficient training or experience is militating against truly early diagnosis. This is particularly true with our present shortcomings in physical diagnosis. This is not said to discourage the making of physical examinations, but in recognition of a fact which is militating against early diagnosis. With the more intensive teaching of today, however, there should be no more excuse for failing to examine chests properly than for failure in any other line.

As long as we are led to believe that early pulmonary tuberculosis is easy to find on physical examination, so long will examinations be made, and, failing to discover a lesion, the patient be told that his lungs are free from tuberculosis. Expert examiners are beginning to realize that the truly early cases cause exceedingly slight changes in the tissues; and that the truly incipient pulmonary tuberculosis—that entity concerning which they have talked and dreamed—is so unfamiliar to them that they would scarcely recognize it when making examinations by the usual physical methods.

The findings on physical examination of moderately advanced tuberculous lesions are fairly well known. They have unfortunately been described as belonging to incipient tuberculosis. This fact can be appreciated when we fully realize that adult pulmonary tuberculosis is a chronic infection; at first, usually small and fibroid in character, because it is an infection which takes place after the patient has become partially immunized against the bacillus. The protective sensitization of the cells prevents it from extending rapidly. Its very slowness of development minimizes the symptoms present; and so the disease usually arrives at such a degree of activity that exudation has taken place in the surrounding tissues, the lesion has become somewhat extensive and conditions are present for a fairly rapid progression of the process before it is recognized clinically. Instead of calling this incipient tuberculosis, it is more appropriately named clinically active tuberculosis. This offers signs which may be detected by the usual methods of physical examination; but even this is not always easy to detect. This process may follow months and even years after the incipient lesion.

The lesson that this impresses upon us is to realize the shortcomings of our older teachings and improve upon them. It emphasizes the fact that signs on percussion and auscultation may be so slight as to go unrecognized, yet a tuberculous lesion be present. Progress is not apt to be made unless the necessity for such is recognized, and it is in that spirit that we approach this difficult subject. If all who examine chests would only appreciate these difficulties, as those do who are expert examiners, and, in case of doubt, make the final diagnosis not so much on the physical findings alone as from a careful analysis of all the data derived from clinical history, physical examinations, tuberculin tests, sputum examinations, and x-ray findings, the results from the standpoint of the patient would be far more satisfactory.

There is no use disputing over the character of the breath sounds in early tuberculosis, for it may be rough; it may be harsh; it may be both; expiration may be prolonged or not. The character of the sound will vary according to the condition of the tissues, whether it be a new lesion or a lighting up of an old focus, and upon the extent of both the old and the new, when present, as described on page 429. The same may be said of percussion. The normal side may show the highest percussion note when the soft structures on the other side have atrophied, as they do in the presence of chronic inflammations in the lungs, as described on page 422.

In case an active clinical pulmonary tuberculosis is suspected from clinical history, and yet the diagnosis is in doubt on account of negative findings on auscultation and percussion, I would suggest that its presence or absence can almost certainly be determined by the presence or absence of the diaphragm reflex (lagging of the side) and the motor reflex (spasm) as it affects the neck and chest muscles, particularly the sternocleidomastoideus, scaleni trapezius, and levator anguli scapulæ, as described on page 410. While these alterations in function may be produced by any inflammatory condition in the lungs, yet they are extremely important in their diagnostic weight when tuberculosis is suspected from other signs and symptoms, and while most of the early symptoms are temporary, these muscle changes (spasm) last as long as the sympathetic endings are irritated by the inflammation in the lung.

An acquaintanceship with the motor and trophic reflex as affecting the muscles of respiration including the diaphragm and the skin and subcutaneous tissue covering the neck and chest muscles, as described on page 405, will afford the examiner most helpful information in these difficult cases.

The position of the sputum examination in the diagnosis of what is usually termed early tuberculosis can be made of far more value than it is. The common methods of taking only a sample, or even telling the patient to clear out his throat mucus in the morning and save that immediately following, are subject to great error. In these early cases bacilli may be present, but they are not apt to be present in all the mucus raised. The surest method of finding them consists in taking a twenty-four hour, or a three day, sample and digesting and homogenizing it as mentioned on page 534. In this way the error is greatly minimized. Sometimes when bacilli are absent a high lymphocyte count may be present (Wolff-Eisner). This is extremely suspicious of tuberculosis. So is the albumin content in the sputum of some diagnostic merit.

The X-ray in the Early Diagnosis of Tuberculosis.—Various opinions are held regarding the value of the x-ray in the diagnosis of early tuberculosis. It is of great value when used by an expert. It must always be remembered that it has serious limitations. No one except an expert should make a diagnosis of clinical tuberculosis from an x-ray plate alone. The findings may be positive, yet the process may be healed. If, however, clinical symptoms are present and physical signs particularly reflex motor disturbance and x-ray findings are positive, then the x-ray is of value in giving important confirmatory evidence. The x-ray as used by the average operator gives no reliable data on which to base a diagnosis of truly incipient tuberculosis. There is no doubt, however, that even a poor x-ray will make the diagnosis earlier than many of the careless clinical examinations. It is on a par with a poor physical examination. The best work in pulmonary röntgenoscopy, however, as studied and compared with the best work of clinicians, the comparison being made by the clinicians themselves, has helped to elucidate many problems and has advanced the science of diagnostics very materially. The x-ray is particularly valuable in hilus lesions; and in this connection I desire especially to emphasize its value in the diagnosis of tuberculosis of the bronchial glands in children. But here the findings are of real importance only as they are taken in connection with the clinical history. For further discussion see Chapter XIX.

The fluoroscopic examination of the chest is important. The study of the motion of the diaphragm will often give valuable information. The patient should be observed both during quiet and forced respiration, remembering that a limited excursion of the diaphragm may show on quiet respiration and be entirely overlooked on deep breathing.

DIFFERENTIAL DIAGNOSIS.

General Asthenic Constitution.—It is often difficult to determine whether or not a person of general asthenic build is also suffering from a tuberculous infection. Such individuals show many of the characteristics of the one with semi-quiescent tuberculosis. The want of energy, lack of endurance, and gradual development of the irritable nervous state, coming on as they so often do in both conditions immediately after puberty, makes them extremely difficult to differentiate at times. If tuberculosis is primarily the cause of the asthenic condition, or if it is present as a complication, it often remains of the hidden type for a long time. Even if localized in the lungs it may not be detected early owing to its slow progress. In the case of hidden tuberculosis the tuberculin test becomes a very important diagnostic measure; and, even it may prove of negative value. Under such conditions a probable diagnosis is all that can be made. If the infection is in the pulmonary tissue, the doubt should be removed by methods already mentioned in the early part of this chapter. The reflex signs and symptoms, and the symptoms due to the tuberculous process *per se* are of far greater diagnostic value than those of toxic origin in these cases, and, when present make the diagnosis easier.

Neurasthenia.—Neurasthenia may be caused by many different conditions, including tuberculosis. At times it offers the same difficulties of diagnosis as the general asthenic condition. Its relationship to the toxemia of tuberculosis must not be lost sight of. A hidden or unsuspected tuberculous lesion is often the underlying cause and should always be considered where the etiology of neurasthenia is vague. See Chapter VI for a more complete description of this relationship.

Malaria.—Malaria is particularly confusing in districts where prevalent. The toxic symptoms of early active tuberculosis so closely resemble those of malaria that it is no wonder that erroneous diagnoses are made. Care on the part of the examiner, however, will differentiate the two, particularly if the lesion be pulmonary. Finding the parasite in the blood settles the diagnosis. One should remember, however, that both may be present.

Acute or Subacute Bronchitis.—The differentiation between simple bronchitis and early pulmonary tuberculosis is at times difficult to make, particularly if the bronchitis is prolonged in its course.

Bronchitis often comes on as a cold in the head or infection of the upper respiratory tract and extends downward, although at times the first symptoms are on the part of the lower tract. Tuberculosis, on the other hand, begins in the lungs. In bronchitis the attack is apt to be more sudden in its onset and the symptoms more severe, although this varies greatly with different attacks. Cough usually appears suddenly and is harassing in bronchitis, while it is of gradual onset and milder in tuberculosis. Temperature is usually elevated at first and drops after a day or two in bronchitis; but it is persistent if due to active tuberculosis; or, if due to a sleeping focus which has been aroused to activity. Râles may be present, either fine, or coarse and bubbling, or dry sonorous, or sibilant in bronchitis, and are, as a rule, scattered over considerable lung area; while in tuberculosis they are confined to a smaller circumscribed area, usually near the apex, and are accompanied by percussion changes and disturbed function on the part of the diaphragm and other respiratory muscles. The motor reflex is not present in bronchitis unless pulmonary tissue is inflamed. The tuberculin test is often of value when sputum is absent. A bronchitis, accompanied by toxic symptoms, which persist for more than a few days, is suspicious of tuberculosis.

Intercostal Neuralgia.—The diagnosis of intercostal neuralgia is often made when tuberculosis of the pleura is the pathological process present. Intercostal neuralgia is an exceedingly rare condition *per se*, while tuberculous pleurisy is common. Judging from the diagnoses made, the opposite would seem true. Any pain which seems like an intercostal neuralgia should be investigated for pleurisy (tuberculosis of the pleura). One important differential point is that the motion of the lung is more restricted in pleurisy than in intercostal neuralgia, and the pain is more acute on respiration. It must be remembered that there can be an inflammation of the pleura without any marked signs. Both pain and the pleural rub may even be absent.

Influenza.—Sometimes clinical tuberculosis is suddenly ushered in with symptoms resembling those of influenza. This usually comes in one who has been somewhat below par for a time, yet in whom the symptoms were so slight that they were not recognized until the more acute symptoms manifested themselves. A chill followed by fever and other toxic symptoms, cough and free expectoration, all within a few days, is the picture. The examination of the sputum in such cases usually affords a surprise to the unsuspecting clinician and makes the diagnosis. The motor reflex as shown in diminished action of the diaphragm and increased tonicity of the neck muscles is present and of value in showing that pulmonary tissue is involved in the inflammation. These attacks are all the more confusing if they come, as they often do, during the season when influenza is prevalent.

Aside from the influenzal type of onset, there is a close resemblance between the usual exacerbation in tuberculosis and influenza. Tuberculous patients often complain of repeated attacks of *la grippe*. Under such circumstances, the very fact of the repeated attacks should arouse suspicion that the process is probably of a tuberculous nature, and cause the sputum to be carefully searched for bacilli, and other means of differentiation to be used.

Chronic Purulent Bronchitis and Bronchiectasis.—Not only are there difficulties in differentiating between early tuberculosis and other lesions in the lung, but even advanced tuberculosis must often be differentiated from other widespread non-bacillary infections.

Chronic bronchitis and bronchiectasis, with the large quantities of sputum which are often present, and the evidence of loss of tissue and cavity formation and the tendency to exacerbation, is often extremely difficult to differentiate from tuberculosis. The difficulty is increased by the fact that chronic tuberculosis may be accompanied by large quantities of sputum, and yet not show bacilli. Cavities, at times, suppurate long after bacilli disappear. On the other hand, cases may be free from bacilli for years and be considered as simple bronchitis and then show bacilli. A patient who had been under the care of a confrere for fourteen years recently consulted me. At no time had bacilli been found in the sputum. Immediately prior to her consulting me, she had a rise of temperature and suffered from malaise and loss of weight. To her great surprise bacilli were found. Three possibilities must be considered in this case; the first, that this was a case of chronic bronchitis which eventually involved tuberculous tissue causing a breaking down of the protective envelope with the escape of bacilli; second, that it was originally a tuberculosis which left the patient with a suppurating cavity from which the bacilli disappeared not to return again until the time of her consulting me; and, third, that bacilli were given off from time to time during active periods without being discovered. Previous examinations had always been confined to the examination of a sample of sputum, while I took a twenty-four hour specimen and submitted it to digestion, and homogenization with the shaking machine before making the slide. I wish to urge the importance of such a procedure in all such cases as being far more satisfactory than the examination of single or repeated specimens. The patient also responded promptly to the cutaneous tuberculin test with a marked reaction, and from this alone I would have felt sure that there was an active tuberculous element in the case. In order to fully understand the necessity of examining twenty-four or seventy-two hour specimens after homogenization, see Chapter XX.

Chronic Fibrosis.—We often find an increased density of tissue over a considerable portion of a lobe, usually the upper, when making a physical examination of the lung. Sometimes it is more marked near, and extending some distance into the lung tissue from the hilus. Auscultation often reveals a diminished murmur at times with increased harshness and sometimes with prolonged expiration. Such a condition may be present for a long time without producing recognizable symptoms, although usually there is a gradual impairment of health.

I am coming to believe that most of such cases are tuberculous in nature from the first, and that they are produced by an organism of low virulence which stimulates to new tissue formation instead of necrosis. Sooner or later, however, these processes undergo necrosis and assume the usual fibro-ulcerative type. The diagnosis in these cases is rarely made until necrosis occurs with the appearance of bacilli in the sputum, although this is usually preceded by a history of general decline in health lasting for a variable time. The x-ray is of value in these cases but is rarely employed unless accidentally, for it is rarely that the lungs are suspected, because there are no particular signs except those of gradual loss of strength until the temperature stage which precedes or attends ulceration is reached.

Pulmonary Infarct.—An infarct of a small pulmonary vessel may at times be difficult to differentiate from a tuberculous lesion. When the source of the embolus is plain it is not so difficult; but, if the seat is hidden, as, for example, in one of the deep veins, doubt may arise. If it is a large pulmonary vessel which is occluded, physical signs may be detected; but on the other hand the vessel may be so small as to preclude any changes in percussion or auscultation. If near the surface, pleurisy is usually present, and motor disturbance on the part of the diaphragm will be noted. Râles may or may not be present according to the size of the infarct. Pain is the most common symptom. It is usually the first one noticed. It resembles the usual pain of pleurisy; which means, that it shows great variability. It may be dull or sudden or sharp in onset; and may, particularly if it affects the diaphragm, be felt in the upper abdomen and shoulder. It may be a slight discomfort or an acute pain brought on by cough and deep breathing. Expectoration of blood is a common symptom and only secondary in importance to pain. The blood may be slight in amount, or very profuse. It rarely shows as bright blood, but usually as tenacious, dark clots. With such symptoms it might be quite difficult to determine whether or not tuberculosis or embolism is the cause. The character of the blood and the previous history of the patient are important factors. The fact that infarcts are more apt to occur in the lower lobes, while tuberculosis usually affects the upper lobes first, is also important. In infarct the symptoms are usually more sudden than in tuberculosis. The fact that infarct usually comes on in patients with cardiac disease or venous thrombosis is especially suggestive. Infarct usually runs a course with slight or no fever, but so does tuberculosis at times. The tuberculin test may give important information.

Pneumonia.—It is a very common experience for physicians, on being called to see a patient for the first time, to make a diagnosis of pneumonia if they find fever present and an altered percussion note over the chest with râles in the underlying lung. This diagnosis is made all too readily. The preceding history should be taken into consideration, the mode of the onset inquired into and the whole symptom-complex should be carefully analyzed before such a diagnosis is made.

Pneumonia, usually, begins with sudden onset, while tuberculosis usually shows a definite history of antecedent decline in health. A careful history alone should nearly always prevent a mistaken diagnosis. For the symptoms of tuberculosis to be so pronounced as to be mistaken for pneumonia, with the exception of those acute exacerbations which simulate influenza, there should be a definite antecedent history showing the presence of some of the most important symptoms extending over a period of weeks and often months. Malaise, lack of endurance, and feelings of gradual loss of strength would usually precede such a widespread infection, while cough and temperature could hardly be expected to be absent. Careful examination should also reveal the fact, if the focus is of tuberculous origin, that it began at or near the apex of the lung; while, if it is pneumonia, the signs are usually in the middle or lower portion of the lung, although either may begin in any part.

Lobar pneumonia is usually readily diagnosed, but bronchopneumonia is the one which offers difficulties and is more often confused with tuberculosis. Physicians seem to dread making a diagnosis of tuberculosis if they can avoid it; the habit is all too common of calling any acute disease in the lungs either bronchitis or pneumonia. The curability of early tuberculosis and the seriousness of late tuberculosis, should be sufficient to compel that the tuberculous patient be given the benefit of the doubt. The examination of the sputum will usually be sufficient, if carefully made, to reveal the bacilli. They are nearly always present when such acute symptoms occur. Repeated examinations should be made if the first are negative. A negative result on examination, however, does not preclude the tuberculous nature of the disease.

If the diagnosis is not made during the acuteness of the illness, a delayed convalescence should make the diagnosis almost certain.

The tuberculin test is unreliable in differentiating these conditions because tuberculosis may not give a reaction during the acuteness of the process. A marked reaction, on the other hand, would give valuable information. The test should be employed, but too much reliance should not be placed on the evidence obtained.

Pulmonary Syphilis.—Pure syphilis of the lung is not a common disease, yet it is one that must be differentiated from tuberculosis at times. It is not unlikely, however, that the two diseases are at times found coincidentally.

It is well to bear in mind that no one has been able to control his ancestors and consequently anyone may be infected with syphilis; and no one has been able to control his environment during early years, consequently anyone may have tuberculosis. These diseases may be associated or they may be found independently. Each of them might infect the lung. Tuberculosis usually affects the lungs in adults; syphilis usually affects nonpulmonary tissues.

Points of differential value in diagnosis are: tuberculosis usually begins at the apex, while syphilis usually begins at the hilus or base. Syphilis elsewhere in the body, with a typical pulmonary infection starting at the hilus or base, unaccompanied by bacillus bearing sputum, forms a basis for the diagnosis of syphilis; so does a positive Wassermann with negative sputum and negative tuberculin tests in atypical lesions. The therapeutic tests are of some value. Prompt improvement under antisyphilitic treatment suggests a syphilitic process.

The symptoms of advanced syphilis may not differ in any particular from those often observed in tuberculosis. This can be appreciated when it is recalled that the symptoms of pulmonary tuberculosis are extremely variable. As a rule, the symptoms are less acute when of syphilitic than when of tuberculous origin for the same amount of pathologic change.

Landis[2] discusses this subject as follows:

"In acquired syphilis most writers describe two main types of the disease as it occurs in the lungs; namely, the formation of gummata or an extensive cellular infiltration which leads to fibroid changes. If the disease assumes the indurative type, and this is by far the most common form, it usually originates at the hilus of the lung and extends outward along the bronchi and blood vessels. The process is usually unilateral, and at most involves only a portion of one lobe; if several lobes are implicated it is the portions which adjoin the root of the lung.

"In addition to these types a focal form has been described in which the lesion consists of an area of consolidation and catarrh. It is usually situated around the root of the lung, and may occur at one apex.

"Whether the disease manifests itself in the form of gummata, as a diffuse fibrosis, or as a focal lesion, most of the cases reported indicate that the base of the lung or the area about the hilus, rather than the apex, is the part most frequently attacked. This fact is usually cited as one of the strong differential points between syphilis and tuberculosis. The general opinion has been that the apices are rarely involved, but it would be rather surprising, in view of the wonderful diverse forms in which the localization of syphilis manifests itself, if the upper portions of the lungs should always escape.

"We believe that pulmonary syphilis of a latent type occurs far more frequently than is usually supposed. The form to which we wish to call attention is that in which the localization occurs in the apices of the lungs. This type of the disease may simulate early pulmonary tuberculosis so closely as to deceive us entirely."

In discussing the diagnosis, he further says:

"It is to be noted that the symptoms in all of these cases were characteristic of pulmonary tuberculosis; namely, morning cough and expectoration, blood-streaked sputum; loss of weight, and a slight elevation of the temperature. In two there was pain resembling that occurring in pleurisy at the base of the right lung.

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[2] Latent Syphilitic Infection of the Lungs, American Journal of Medical Sciences, August, 1915.

Furthermore, they all had physical signs indicative of incipient tuberculosis.

"If the lungs are involved, there is, in addition to the symptoms mentioned above, cough, which may be dry and unproductive or accompanied by a moderate amount of greenish or yellowish expectoration. Blood-streaked sputum may also occur. Pain at the base of the right lung is not infrequent and may be misinterpreted; it is usually due to a syphilitic perihepatitis and not to pleural inflammation.

"The presence of latent syphilis of the lung is to be suspected if in addition to pulmonary symptoms there are present elsewhere in the body lesions which are in all probability luetic in nature, such as a periostitis, orchitis, iritis, or suspicious throat lesions. The recognition of these cases should be relatively easy.

"The type of the disease which offers the most difficulty, and which, for the most part, escapes detection, is that in which the symptoms are entirely pulmonary and in which there are no associated syphilitic lesions.

"The diagnosis must be made by exclusion. Thus if the symptoms and physical signs are those characteristic of tuberculosis, and the sputum does not contain tubercle bacilli, or the progress of the case differs from that usually encountered in tuberculosis, the possibility of some other exciting cause should be thought of. Not only should the sputum be examined for organisms other than the tubercle bacillus, but in addition, a Wassermann test should be made in every doubtful case."

Actinomycosis.—This affection must sometimes be differentiated from tuberculosis. The differentiation depends on finding actinomyces in the sputum. In symptoms and signs the disease is much the same as many of the atypical cases of tuberculosis. Cough, sputum, at times accompanied by blood, dyspnea, night sweats, and failing strength are the usual symptoms. The sputum was very fetid in one of my cases, while in another it did not differ especially from that of tuberculosis.

This affection is often found at the bases instead of the apices, although it may affect the latter.

Negative tuberculin reactions and a failure to find bacilli are only negative evidence. They do not afford any positive diagnostic data. The finding of actinomyces is necessary for diagnosis.

Streptothricosis, Blastomycosis, Aspergillosis, and Coccidioidal Granuloma are sometimes to be differentiated from tuberculosis. These diseases are rare but should be thought of when the pulmonary lesion is atypical. In comparison with tuberculosis they are so rare that they sink into insignificance; but, for accuracy of diagnosis, they must be borne in mind. Their diagnosis is made by finding the specific microorganisms in the sputum.

Malignant Tumors of the Lung.—Malignant tumors of the lung may be either primary or secondary. Such primary growths, however, are comparatively rare. They may be either of the sarcomatous or carcinomatous type, the former occurring in early life, the latter in late life. Probably two-thirds of sarcomata occur before the fortieth year, and nearly all carcinomata after that time.

Of ten cases of sarcoma and cancer of the lung seen by me, and of which I have notes, all were secondary but one. This was a primary cancer, taking its origin from the glands in the left superior bronchus. The others were metastatic; one a carcinoma primary in the mediastinum; two, primary in the stomach; and three, primary in the breast. The other three were sarcomata; two, primary in the femur; and one, primary in the uterus. In one of the sarcomata the leg had been amputated for sarcoma nine years previous to the metastatic manifestations.

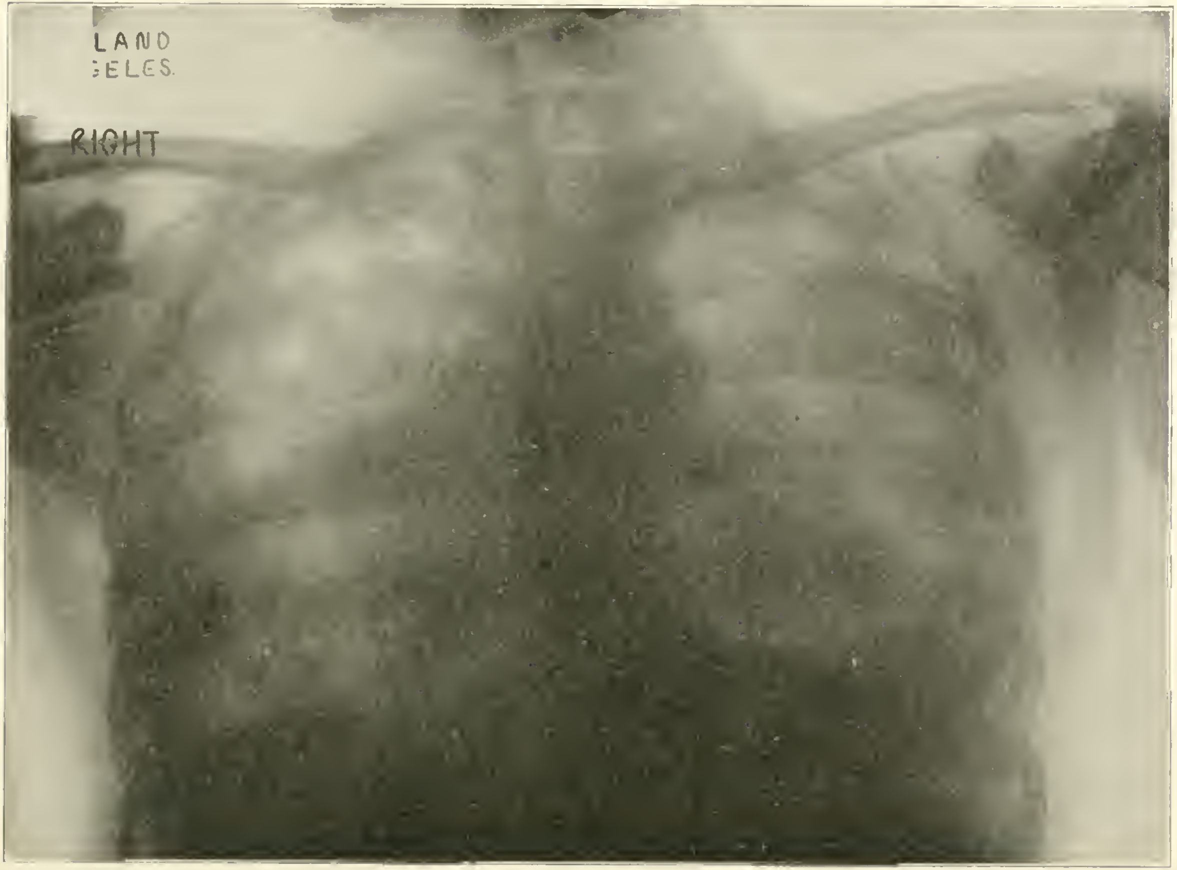
These growths may extend throughout more or less of the lung, and also affect the pleura and structures of the mediastinum. The pericardium is not uncommonly affected. Pleural effusion is not uncommon and adds greatly to the discomfort of the patient, particularly late in the clinical course of the disease.

The symptoms vary greatly in different cases according to the structures involved. An irritating *cough* which slowly increases in severity is common. This is at first unproductive or accompanied by a small quantity of glary, pearly, or, at times, greenish mucus; the latter being found rarely except when the disease has become somewhat extensive. *Dyspnea* of a progressive nature is also common. This is especially emphasized on exertion. The patient may be comfortable when quiet, but, on changing position, suffers from shortness of breath. *Temperature*, is, as a rule, not present early. I have seen a rise to 100° however, as the disease became advanced and showed signs of breaking down. *Pain* is, at times, a factor. In the case of my series, which was primary in the mediastinum, there was marked pain under the sternum and in the left arm. A very marked involvement was present in the pericardium in this case. In some of the other cases, sharp cutting pain like that of acute pleuritis was present; and, in two, considerable dull pain in the axillary region. A feeling of fullness and compression is particularly noticed where the growth spreads rapidly and where large pleural effusions are present. *Hoarseness* and *aphonia* may appear when the recurrent nerve is directly involved. Difficult breathing or *stridor* is sometimes noted when pressure is made on the trachea.

In differentiating cancer and sarcoma from tuberculosis it should be borne in mind that the toxic and most of the reflex symptoms present in the latter disease, are absent; and that, in cancer and sarcoma, the process in the lung is usually widespread for the clinical symptoms present. The patient may be in full weight until near the time that the process becomes fatal. On the other hand, I have seen cachexia and emaciation the same as in pulmonary tuberculosis.

The physical signs present depend partly on where the growth is situated. In the centrally located growths there is increased resistance on palpation and percussion, as well as a dullness over the areas surrounding the hilus of the lung; while in pure pulmonary metastases there are either isolated or conglomerate tumor masses in the pulmonary tissue itself which if large enough produce the same changes.

The side of involvement usually shows diminished motion. When located in the mediastinum pressure on the vessels at times becomes great and causes a dilatation of these on the surface. Auscultation may or may not show râles. Diminished breathing may be present when pressure is made by the tumor upon a bronchus and when air is excluded from the pulmonary tissue by the tumor structure. The supraclavicular, cervical and axillary glands may be the seat of metastatic infection.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The sputum may be blood-tinged at times. Sometimes fragments of the tumor are found, and sometimes clusters of cells which are considered as having diagnostic importance.

The x-ray is of value where its findings are correlated with that of physical examination and clinical history. Fig. 105 shows the x-ray plate of one of my cases.

Diagnosis of primary cancer in the lung is not easy. In my case, the widespread lesion, with little or no sputum, comfort during quiet, with dyspnea on movement, absence of temperature, and a peculiar feeling to the palpating fingers which was entirely different from tuberculosis caused me to make a diagnosis of probable new growth in spite of the absence of a history of cancer in some other portion of the body. Later, tumor cells were found in the sputum and the case was proved postmortem.

In the secondary metastatic tumors of the lung, the history of a previous tumor in some part of the body is suggestive.

CHAPTER XXII.

PROGNOSIS.

Introductory Remarks.—There are many factors which must be taken into account in arriving at a prognosis, each one of which influences the chances for recovery, to a greater or lesser degree.

It is but a few years since tuberculosis was considered an incurable disease. Today, however, clinical experience warrants the assertion that tuberculosis is not only curable, but that, when treated at the right time, and in the proper manner, it may be overcome and the patient be restored to health in a very large per cent of cases. Early clinical tuberculosis when properly treated, should have a mortality no greater than that of other common infectious diseases.

The hopeful side of the tuberculosis problem has been well stated by Elsner.[1] In speaking of chronic tuberculosis, he says:

"A large number of these cases are curable, many become latent; the vital fact which interests us in considering their future is that they can only be saved by *early diagnosis and prompt treatment. Put in the resistance, bring the condition of the patient to par or above, and the disease, if it is in its incipiency, will be stayed in over eighty per cent of all cases, while twenty per cent of all forms of the disease are saved. To wait for definite physical signs before making a diagnosis darkens prognosis, for the patient's chances are reduced thereby. To anticipate the final development in cases which are strongly suggestive, adds to the patient's chances. Positive physical signs are never early evidence of lung infection; they mean that the case is advanced.*"

Age.—The prognosis differs according to the age of the patient. The most serious age period for tuberculosis, all things else being equal, is the first five years after birth. During this period the patient is gradually developing a specific cellular resistance, and, until such time as this has been attained, the organism is not capable of overcoming large quantities of tubercle bacilli; consequently, if a severe infection occurs during this period, the patient has little chance of overcoming it. As shown by the statistics of Hamburger, quoted in Chapter IV, there is practically no tendency toward healing shown in patients suffering from tuberculosis during the first two years of life; and it is only after the fifth or sixth year that the child begins to show an increased resistance. From that time on, we see more of a tendency for the disease to become chronic. By the time the child reaches the fifteenth year chronicity is manifested in one-half of the cases; and, after the fifteenth year, chronicity becomes more and more common; in fact, becomes the rule.

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[1] Monographic Medicine, D. Appleton & Co., 1916, vol. vi.

Constitution.—While we have learned in recent years that tuberculosis is not an inherited disease, yet it is necessary for the clinician to take into consideration the constitution which is given to the individual at birth. While environment is probably a much greater factor in the development of a child than heredity, yet there is no question but that heredity counts for something. The inheritance of a weakly constitution in an individual who lives in a poor environment, makes the worst combination possible. There are certain people who seem to be born with deficient nervous and physical mechanisms. Stiller has called attention to one type, *asthenia congenita universalis.* This type, he believes, is born with nervous and physical mechanisms below par, even though the asthenic state does not develop until after puberty. Many individuals in whom a weakly constitution has been recognized in the past, do not inherit this constitution, but develop it after birth; in fact, many of those who are physically below par, and who are suffering from so-called *phthisicus habitus,* are not fit subjects for infection, as is usually expressed, but have this habitus because of a previous infection. A person with a weakly constitution, naturally, should not be expected to have as much resistance to disease as one who is physically and nervously more fit; at the same time, it does not mean that those with weakly constitutions must be given up as hopeless, should they develop tuberculosis.

It has been my lot to treat a great many patients with poor physique, who had developed tuberculosis; and, in case the infection was not too severe, I have seen excellent results obtained in many of low physical and nervous vigor.

Environment.—The environment in which the patient lives probably has more to do with prognosis than any other factor. This term "environment" means so much that it comprises many other subjects which will be discussed in this chapter. I shall discuss environment, however, giving to it a limited meaning.

If we discuss environment in connection with the physical surroundings of the patient we can see what an important element it is in prognosis. It further includes the home surroundings, in which the patient lives, as well as his social and economic status. All of these are big factors in determining whether or not a patient with tuberculosis shall get well. Environment may be helpful, as well as harmful. If a patient lives in a stuffy room, with dead air, his opportunity for recovery is not as good as though he were in the open air. If he is surrounded by depressive circumstances, and depressive conditions, naturally, it will affect prognosis unfavorably. If the members of his family, and the friends with whom he lives, are cheerful and happy, and determined to aid him in carrying out his routine, his chances for recovery are far greater than where they are opposed to such a routine. It is possible to surround a patient in a home with cheerfulness and hope and give him encouragement, and so arrange the physical features of the home as to aid; or they may be arranged so as to have the contrary effect. Good helpful environment adds greatly to the patient's chances of cure; while antagonistic depressive environment makes the prognosis much less favorable.

Economic Status.—Tuberculosis is an economic disease. It is found much more commonly among the poor than among the well-to-do, but exists in people in all stations of life.

Now that we understand tuberculosis to be a disease which is transmitted through ignorance and willfulness, and, since infection takes place largely in early life, and clinical tuberculosis develops from this early infection, it can be readily understood that the economic status of the individual is a big factor in prognosis. Ignorance and poverty go together, yet we find many worthy intelligent people among the poor, and find some of the grossest ignorance among the well-to-do. Poverty compels people to live under unsanitary conditions. It is necessarily accompanied by an inadequate amount of food; and it deprives them of many of the healthful pleasures of life. Thus it acts by producing a general depression of the physical and mental capabilities of the patient.

In those homes of the poor, in which overcrowding occurs, there is great danger of the disease being communicated to others; and, inasmuch as lowered vitality favors infection, this also operates to make transmission easy. Continued living under such circumstances makes those individuals who are already infected, more prone to have an extension or recurrence of their disease.

We must conceive that tuberculosis is a disease which, taken into the body early, makes more or less effort in after years to extend to other tissues and parts. If the patient's resisting powers are low and his body cells are impoverished, they are not able to react with the same defensive force as the cells of those who are in better physical and nervous condition; consequently, the economic status of the people is a great factor in providing for their material wants, hence an important factor in the prognosis of tuberculosis. It is also important from the standpoint of treatment. Unless the state has provided adequate care for those who are ill, those whose economic status is low are unable to cope with the disease. Tuberculosis is a long drawn out disease, the individual being an invalid and unable to work for a time averaging about two years. During all this time he must not only be cared for; but, if he is the bread winner, the remaining members of his family must also be cared for or suffer; consequently, if poverty is present at the beginning of the disease, it will grow worse as it continues.

The state and the municipality have both begun to see their duty toward the tuberculous poor, and we now have provision made for many of those who are ill. Such provision, however, is still inadequate, and must be supplemented by private philanthropy to as great an extent as possible until full provision is made for all.

While the financial condition of the patient is an important factor, yet this does not imply that the prognosis in the wealthy is better than in those of lesser means. On the contrary, it is often rendered unfavorable by other conditions which come with the possession of large means. It has been my observation that the best patients to treat are those of the middle class. Here we have intelligence, combined with enough means to care for the patient, yet we do not have to fight the ignorance which is so common among the very poor, and the willfulness which is so common among the very wealthy.

Mental State.—The mental state of the patient is very important in prognosis. Now that we understand the action of the depressing emotions and other psychical states in keeping up a prolonged stimulation of the vegetative nervous system, and, through it, a disturbance in function on the part of many of the important internal viscera, we can offer a physiological explanation for a fact which has long been known; namely, that prognosis depends very much upon the mental state of the patient. The patient will often worry about some symptom which is comparatively unimportant. He will complain and become angry over the failure to have some little whim satisfied, when his very dissatisfaction and anger will do more harm than the conditions about which he was complaining. From a prognostic standpoint, hope, cheerfulness, and contentment are very important.

THE CHARACTER OF THE LESION IN PULMONARY TUBERCULOSIS.

Much could be written upon the subject of the character of the lesion in tuberculosis, but I will attempt to discuss it briefly from a practical standpoint.

Miliary Tuberculosis.—Miliary tuberculosis is usually spoken of as being fatal. This is not true. In generalized miliary tuberculosis this is true; but, in all cases of tuberculosis that are at all extensive, we have areas of miliary infection, which yield to the defensive powers of the patient. Miliary tuberculosis must not be confused with acute general miliary tuberculosis. The former refers to the small tubercles, while the latter refers to a dissemination of the disease more or less generally throughout the body, the infecting bacilli coming from a common focus and becoming implanted in the tissues at the same time. The former is favorable for cure; the latter practically hopeless.

Fibroid Tuberculosis.—Now that we have studied more of the history of our infections in tuberculosis, and realize that they start in childhood, and gradually extend by secondary metastases, we have learned that infiltrations are often present for years without producing open active tuberculosis; but we are further coming to learn that there are types of infection which are comparatively harmless during their early stage, but which take upon themselves ulceration and become serious as a later manifestation. Not infrequently do we see lungs in which a gradual extension of the disease from one focus to another has been going on over a prolonged period of time. Such patients, as a rule, show evidence of a chronic toxemia. They have a deficient nerve balance; unstable heart's action; suffer from stomach disturbance, with a resultant malnutrition; and show a disinclination and inability to carry on work. They may have a slight rise in temperature at times, or they may not. Finally, after this stage of semi-invalidism has gone on for a period of time,—it may be a few years or it may be many years,—the patient is surprised by some such symptom as hemorrhage, pleurisy, repeated attacks of bronchitis, and probably expectoration which shows bacilli.

Did we recognize this form of tuberculosis early, nearly every one of these patients could get well; but, as long as we delay our diagnosis until the more active symptoms manifest themselves, just so long will we fail to do our duty by these patients by treating them at a time when the prognosis is favorable. We should bear in mind that chronic fibroid tuberculosis has as its terminal picture, in most instances, chronic fibro-ulcerative tuberculosis; consequently it demands early recognition and early treatment if it is to offer a favorable prognosis.

Chronic Ulcerative Tuberculosis.—This type of tuberculosis in a pure form is rarely, if ever, found, because all chronic tuberculosis must be accompanied by some fibrosis. What I refer to, however, is the type of tuberculosis in which the ulcerative form predominates. We often see a patient who has had a previous infection in the lung, suddenly manifest acute symptoms with ulceration and cavity formation, and bacillus bearing sputum. From the first ulcerations, bacilli escape and form metastases; and repeated metastases follow necrosis and caseation of other areas. If this type of disease manifests a tendency to heal, it will sooner or later take upon itself a combination of the ulcerative and fibroid form. Areas will cease becoming necrotic and the stimulation of the fixed cells will result in the increase of fibrous tissue with a tendency to heal.

Acute Caseous Tuberculosis.—This is the type of tuberculosis that we often see where there is a large area,—often a lobe, or more commonly, a portion of a lobe, in which the disease develops rapidly going on to necrosis. The infection in this type of disease often comes from plugging a bronchus with bacillus-bearing sputum. The infection is virulent because of the fact that a great many bacilli are implanted at the same time, and overcome the patient's resisting power. The entire tuberculous area is in the same degree of pathological change. This type is accompanied by a severe toxemia, which results in a breaking down of the patient's defense; consequently, the disease usually develops rapidly.

This type of tuberculosis sometimes offers a favorable prognosis. If widespread, however, as a rule, the toxemia is so severe that it proves fatal. I have often seen a caseous pneumonia involving an entire lobe come to a state of arrestment. It did this by practically amputating the entire area of infection. Necrosis and caseation occurred and the entire mass sloughed out, leaving an empty shell. In such cases, if the process does not last too long, and if the tissues can slough out within a reasonable time before the patient's vitality is undermined, an arrestment may result. If, on the other hand, the toxemia continues until the patient's metabolism is seriously interfered with, but one result can be expected,—a fatal termination.

While there are many other forms of pulmonary tuberculosis that might be mentioned, yet these are the common types that are met with in every day practice. From the prognostic standpoint, the small lesion is most favorable. The small lesion is nearly always fibroid in character. It is accompanied by little or no collateral inflammation, and little or no necrosis and caseation. It is much easier to attain a favorable result in the lesion which is small and of such a character that the danger of scattering the infection is comparatively remote than in the extensive active one.

TUBERCULOUS COMPLICATIONS.

Not only must we consider the type of the pulmonary disease, but we must consider the complications which are present. It is possible to have a primary infection of almost any tissue or organ of the body, as I have discussed in the chapters dealing with the various tuberculous lesions; nevertheless, nearly all infections outside of the lymphatic tissues are metastatic in character and must be looked upon as taking place after an infection has already existed in the body, and after the cells have taken upon themselves the property of producing defensive substances against the bacilli. Nearly all such lesions heal or become quiescent at first, because the number of bacilli producing them is small, and the infiltration is not extensive. The prognosis, therefore, should be good, unless the infection is localized in tissue of such a character as to produce serious or fatal symptoms.

Tuberculosis of the Larynx.—Tuberculosis of the larynx, in my experience, has always been secondary to an infection somewhere else in the body,—usually the lung. The likelihood of a primary involvement of this organ is extremely remote. The infection is usually a surface infection, taking place from the bacillus-bearing sputum, as it comes from the ulceration in the lung. The prognosis of tuberculosis of the larynx depends both upon the character of the lesion in the lung and that in the larynx. The prognosis, barring those cases which are so severe as to interfere with nutrition, is about the same as that of the accompanying pulmonary disease. If the lung is actively inflamed and undergoing a process of rapid necrosis and caseation, there is little chance of the tuberculous infection in the larynx healing, no matter how slight its character. On the other hand, if the tuberculosis in the lung is arrested, or only moderately active, even severe ulcerations in the larynx may heal.

If we speak of the laryngeal involvement as a process by itself, it may be said, the same as in tuberculosis of the lungs, that the prognosis is in proportion to the severity of the lesion. Slight infiltrations will nearly always heal if the condition of the patient be fairly good and the pulmonary condition be not too extensive nor too active; moderately advanced lesions will heal in a very large percentage of cases under similar circumstances; and severe lesions will heal occasionally if the lesion is located so that the patient can maintain his nutrition.

Tuberculosis of Intestines.—Tuberculosis of the intestine is generally looked upon as offering an unfavorable prognosis. This is not necessarily true, although it must be considered a grave complication. A limited involvement of the intestine may be present for a prolonged period of time without producing serious symptoms, or without seriously interfering with the nutrition of the patient. Under such circumstances healing occasionally occurs. I do not doubt that in many instances infiltrations in the bowel heal without their presence having been suspected. When the lesion becomes extensive, however, then digestion and assimilation is interfered with to such an extent that the prognosis is grave.

I have a patient under my care at the present time who has had a tuberculous involvement for a year and a half or two years, the exact time I do not know. It was discovered at operation eighteen months ago. At that time ulcerations were present from the pylorus to the descending colon. This patient has never had a diarrhea; neither has she suffered markedly with gas pains. She has, however, had difficulty in maintaining her nutrition. She has gradually lost weight and strength.

One of the serious factors in tuberculosis of the bowel, from the prognostic standpoint is the fact that opportunity for repeated surface infection is always present, and even though the part may be removed, the same likelihood of recurrence is present as existed prior to the operation.

Pleurisy.—Pleurisy often causes an early diagnosis of tuberculosis to be made and in this way causes the patient to secure therapeutic aid at a time when the prognosis is good. Pleurisy itself also has a definite influence on prognosis at times. The average pleural adhesions at the apex probably make very little difference, as far as the patient is concerned, except as they cause intermittent intercostal pain and interfere slightly with the inspiratory act; but a pleurisy with effusion at the base, which fails to absorb, and results in a general fibrosis involving the lung, proves decidedly unfavorable. So, at times, do widespread adhesions. Adhesions extending over the entire lung interfere greatly with the inspiratory act and in this way favor splanchnic congestion, interfering with the return flow of blood to the heart. The general venous congestion which shows itself when widespread pleural adhesions are present, must also interfere with nutrition because perfect metabolism depends upon the normal rapidity of blood flow.

Pneumothorax.—Pneumothorax, strictly speaking, is not a tuberculous complication, yet it may be regarded as such because of the fact that it results so often from a rupture of a tubercle in the visceral pleura. Spontaneous pneumothorax is often looked upon as being an extremely serious complication in tuberculosis. This has not been my experience. I have had favorable experiences following its appearance more often than otherwise. In the majority of cases that have come under my care, the patient has not only recovered temporarily, but continued to make normal improvement after the complication had passed away.

Tuberculous Meningitis.—Now and then a case of tuberculous meningitis is reported as having healed. There is no reason why this should not be true. A small localized tuberculous involvement in the meninges might exist without producing symptoms of sufficient gravity to prove serious. In case the patient's resisting power is good there is no reason why healing should not occur now and then in small lesions. When the disease has become extensive and widespread, however, healing is beyond question. Meningitis is usually accompanied by an exudative process which produces pressure, resulting in deleterious symptoms, and, sooner or later, death.

Bacilli.—The presence or absence of bacilli in the sputum greatly influences the prognosis in tuberculosis. Recently, a sanitarian of note, when giving a public address, made a statement which gave rise to the inference that patients with tubercle bacilli in their sputum are in a hopeless stage of tuberculosis. What he probably meant was, that as physicians, we have lost time and reduced the favorableness of the prognosis very materially by not making diagnoses earlier; but, it would be wholly wrong to let the impression go out that patients who have tubercle bacilli in their sputum cannot get well. There is no doubt of the difficulties which are met in endeavoring to bring about healing in patients who are suffering from open tuberculosis; but a large per cent of those who have but recently entered upon the open stage of the disease, with tubercle bacilli in their sputum, can get well if given proper treatment. The difference between the closed focus and open tuberculosis, however, from the prognostic standpoint, is a long step. The prognosis drops very rapidly after ulceration and bacillus-bearing sputum appears. There are two reasons for this: One, the increased danger of infecting other areas through the bronchi; the other, the increased difficulty of healing a broken-down necrotic area.

The form of the tubercle bacillus has also been spoken of as having diagnostic significance. In our work in the Pottenger Sanatorium, after making careful examinations of sputum for many years with reference to the morphology of the tubercle bacillus, we have confirmed the idea suggested by Sewall many years ago, that the short bacillus accompanies activity, and the long bacillus is a representative of chronicity. This does not mean, however, that patients with short bacilli in their sputum cannot get well. We often notice an increase in the relative numbers of short, as compared with long bacilli, even in patients whose progress is favorable. It must be remembered that the lungs of patients suffering from advanced tuberculosis represent many different areas of infection, which differ in age. There are recent ulcerations, ulcerations moderately old, and ulcerations which have existed sometimes for months and years. The more recent ones probably throw off a preponderance of short bacilli, and the chronic ones a preponderance of long bacilli; consequently, we must expect a mixture of the various types; and, when we recall that the chronic ulceration had to go through the acute stage, so may we expect these acute ulcerations to pass on to the chronic stage and produce the long type of bacillus. For further discussion of this subject see page 564.

The Tuberculin Reaction.—Much has been written regarding the prognostic importance of the reaction to tuberculin, particularly the cutaneous and conjunctival reactions. Wolff-Eisner, during his many studies of the tuberculin reaction, claimed that the patient who reacted well to the conjunctival reaction offered a good prognosis. Others have claimed that a marked cutaneous reaction is prognostically favorable. This question cannot be discussed simply as a matter of reaction because it is necessary to take so many other things into consideration.

I, personally, have always interpreted a good local reaction at the point of injection as giving evidence of the presence of specific defensive substances. I have noted that the patients whom I have treated (I usually employ an emulsion) usually show an increased tendency to local reaction as their disease improves. Some patients, however, make a perfectly satisfactory improvement and do not show these local reactions.

There are many things to be considered in interpreting tuberculin reactions. Sometimes the tuberculin is absorbed and carried off readily. Under such circumstances we would not expect to have a strong local reaction. At other times, it will remain in the tissues and meet the specific enzymes which are prepared for its destruction, producing a local reaction, which is more or less marked. If the tuberculin reaction is an antigen-antibody reaction, whether it be local, or focal, then it must be taken as evidence of the presence of a specific defense. It must not be considered, however, that the patient who shows this specific defense will necessarily get well of his tuberculosis. This depends very much upon the future course of the disease, as well as upon the way the case is handled. Such defense may change from day to day, from week to week, or from month to month; consequently, it is very difficult to discuss this question as an abstract proposition. It must be considered only at the time of its exhibition, and must be expected to change as the disease progresses.

NON-TUBERCULOUS COMPLICATIONS.

Non-tuberculous complications may arise on the part of any organ or structure. The ones which require particular consideration, however, are those which confine themselves to the four great systems of the body, the nervous, respiratory, circulatory, and digestive.

Nervous System.—On the part of the nervous system we have numerous complications which bear upon prognosis. Nearly all patients who suffer from tuberculous toxemia have disturbances on the part of the cells of the central nervous system. This results in more or less depression of nerve tone. Neurasthenia is a very common complication of tuberculosis; in fact, nearly all patients have it to some degree. It influences prognosis to the extent that the neurasthenic symptoms are permitted to interfere with the patient's metabolic activity. A neurasthenia of mild degree does not seem to have a particularly antagonistic effect; while, in some cases, a severe degree may have an influence in making the prognosis unfavorable secondary only to the tuberculous process itself. All of the depressive nervous states produce a general inhibition of the functions of the enteral viscera and in this way influence prognosis unfavorably.

Not only do we have a general loss of nerve tone designated as neurasthenia, but we have an actual degeneration of nerve cells as a result of the tuberculous toxemia. This is sometimes sufficiently extensive to produce definite organic changes in the cells of the central nervous system. This is not infrequently found postmortem in subjects who have had a prolonged toxemia.

Aside from these disturbances we have the effect of the tubercle toxins influencing the nervous and endocrine systems, and producing changes in the function of the internal viscera. Inasmuch as the nervous system largely controls cellular activity, being supplemented so far as we know, only by the chemical control which comes from the various internal secretions, we can see how important a factor in prognosis the nervous system becomes.

The Respiratory System.—In our discussion we are speaking mainly of pulmonary tuberculosis. Aside from the lesion in the lung or larynx, should there be a complication on the part of that organ, we have other disturbances in the respiratory tract of a non-tuberculous nature which affect prognosis. We might mention the bronchitis which is often present, and the malformations of the upper air passages, as well as the acute infections, such as influenza and pneumonia, which commonly occur during the process of the disease. All of these affect the prognosis to a certain extent.

There is considerable discussion whether or not the patient should be operated upon for conditions which interfere with normal nasal respiration, during the time that he is suffering from active tuberculosis. Personally, I have always avoided operations during active disease, and feel that it is better to spare the patient the lowering of vitality which might follow the operation. The after-effect, however, should normal respiration be restored, might overcome any deleterious influences of the operation itself. I would not counsel operation, however, during periods of activity, but confine it to the times when the disease is quiescent.

An attack of influenza superimposed upon tuberculosis is often disastrous. In many instances, the death of the patient can be traced to a severe attack of this disease. Pneumonia also oftentimes proves serious for the tuberculous patient; at the same time, I have seen patients with a widespread tuberculosis go through attacks of influenza and pneumonia without apparent harm resulting.

The Digestive System.—The non-tuberculous complications on the part of the digestive system have a very important bearing upon the prognosis in tuberculosis. Good nutrition is essential to cure. The tuberculous patient is particularly prone to have disturbances on the part of the gastrointestinal tract, as described in Chapter X. The outcome of the case will often depend upon the success with which these complications are handled. Sometimes what appears to be a very serious interference with digestion may be overcome by proper treatment and in this way an unfavorable prognosis be averted.

The clinician should always bear in mind the influences which are operating to interfere with the function of the gastrointestinal tract. Toxemia always tends to decrease the gastrointestinal secretions, as well as interfere with the motor function of the stomach and gut; consequently, toxemia should be relieved as quickly as possible. Depression has the same effect upon the gastrointestinal tract. This should be relieved by putting the patient in the proper frame of mind. There are reflex symptoms on the part of the stomach and intestines which result from the inflammation in the lung. These may have a tendency to cause a hyper- or hypo-secretion of either the stomach or intestinal glands. They may also have a tendency to increase or decrease motility in both the stomach and intestines. By careful analysis, the exact condition may be arrived at and the disturbance be corrected. While the tuberculous patient may have gastric ulcer, duodenal ulcer, colitis, appendicitis, or any other complication which affects the gastrointestinal canal, yet the majority of disturbances on the part of the digestive tract are of a functional nature and come through disturbed equilibrium on the part of the vegetative nervous system.

The Circulatory System is greatly disturbed in tuberculosis, as mentioned in Chapter IX. A deficient inspiratory act interferes with the normal balance between the arterial and venous system, causing a storing up of blood in the veins, particularly in those of the splanchnic area, and a relative decrease in the amount contained in the arteries. Because of the disease in the lung, many vessels are obliterated, and the right heart finds itself compelled to work against an ever increasing pressure. As a result of this, hypertrophy of the right ventricle eventually takes place. As a result of toxemia and malnutrition, the entire body cells degenerate as the disease progresses. The heart muscle itself becomes less able to do its work and atrophies.

The final result obtained in tuberculosis depends largely upon the integrity of the heart. In those cases where an arrestment of the disease has been obtained, which is accompanied by widespread fibrosis, the patient, as a rule, lives as long as the heart will stand the strain. The patient with an advanced destructive lesion is, as a rule, living on the reserve energy of the heart; consequently, it is unable to measure up to increased demands made upon it. The prognosis in tuberculosis depends very much upon the heart muscle and the proper movement of the blood; for upon these depend the ultimate metabolic activities of the body.

Urine.—The urine during tuberculosis shows several important variations from the normal. If the kidney is involved in the tuberculous process, we are apt to have at first a polyuria; and if the calyx is involved, this is usually followed by pus, and blood appearing later. Where an amyloid degeneration of the kidney takes place it is not uncommon to find albumin. Amyloid degeneration is a disease which affects the blood vessels and is usually progressive. There may at first be a small area involved which permits of the escape of albumin into the urine. As the process extends, the blood vessels involved become destroyed and the urine may lose all traces of albumin for a time. It may be free over a prolonged period until the process, becoming more extensive, causes a continuous albuminuria. This being a waxy degeneration of the blood vessels, and usually being associated with the same process in other internal viscera, usually offers a bad prognosis. It means a progressive pathology which will, sooner or later, lead to the death of the patient.

The Diazo and Urochromogen Reactions.—These reactions frequently make their appearance in the urine, particularly in advanced patients, who are progressively losing ground. These reactions, if continuous over several months, as a rule, suggest a bad prognosis. Tuberculous patients often, however, will have these reactions appear during an acute illness. Under such conditions they disappear as soon as the acute illness is over, and are not prognostically bad. When these reactions come, however, and persist for several months, the chances of the patient's body cells ever being able to carry on their normal physiological function again is highly improbable. (See Chapter XX, page 569.)

Blood in Tuberculosis.—The condition of the blood in tuberculosis has prognostic significance. It is self-evident that the blood which is nearest normal in its constituent parts, functionates the most perfectly. Inasmuch as the blood is the carrier of oxygen and food to the cells, and of the broken-down products to the excretory organs, the necessity of preserving its normal carrying power is self-evident. This is particularly true in tuberculosis, because we have so many other factors which also go to interfere with metabolic activity. The circulatory system is disturbed so that the blood does not circulate freely and normally. We have interference with the normal oxygenation of the blood. We also have degeneration in the various cells of the body so that they are not able to functionate normally, even though the blood is able to supply their needs and relieve them of their effete prod ucts with its usual efficiency. From this it will be seen t the condition of the blood has a very important prognostic bearing in tuberculosis, and it should be our endeavor to keep it standard as high as possible.

Pregnancy.—Pregnancy influences prognosis in clinical tuberculosis unfavorably. Many years ago it was noted that certain patients suffering from clinical tuberculosis improved during the pregnant term, and it was not uncommon for physicians to advise tuberculous women to become pregnant as a therapeutic measure. This observation, however, like many others in medicine, was not complete. While some women improved during pregnancy, many became more seriously ill and many of those who showed improvement during the term lost all that they had gained and suffered from a more active disease after delivery. I look upon pregnancy as being one of the most serious complications in active tuberculosis. The extra burden upon the patient during the period of gestation usually results in reducing her vitality and permitting the disease to extend. Then, the strain of delivery, and the extra cares and burdens which result in the care of the child, are almost sure to bring about increased activity of the disease.

The question of whether a woman who has suffered from active tuberculosis may safely bear a child is one that must be carefully considered. This question cannot be answered in the abstract, but conditions in each individual case must be carefully considered. A woman of the middle, or well-to-do classes, who could be properly cared for during gestation, delivery, and after delivery, would be running less risk than the poor woman who could not have such care. As a rule, I advise tuberculous women who have suffered from an early active tuberculosis, that it is unwise to bear children until they have been well for a period of at least two years. If they have not shown any active symptoms for two years' time, and, providing they can have the proper care and attention, I then feel that the danger of child bearing is comparatively slight. The burden of childbirth is often less than the mental effect of feeling that they are not able to bear children. The danger of lighting up a process which has been quiescent is relatively greater in proportion to the severity of the disease. Many advanced and far advanced cases, under no circumstances, should be advised or permitted to undergo the strain of childbirth.

The question arises, should a woman suffering from active tuberculosis, or one who has recently secured an arrestment, become pregnant, what is the proper course for her safety? This is another question that must be considered individually. In many cases it is wiser to interrupt the course of pregnancy, if it is diagnosed early. Other cases may be properly cared for and the pregnancy be allowed to continue. Under all such circumstances, however, if the pregnancy is allowed to continue the patient must receive careful attention, and must not be allowed to go through a long strenuous delivery, and must be given every care possible following the birth of the child.

PROGNOSIS DEPENDS ON CHARACTER OF TREATMENT.

Prognosis From the Standpoint of the Physician.—The prognosis in a given case of tuberculosis depends very much upon the physician who treats the case. This is a disease which has a tendency to heal; but, at the same time, in a large majority of cases, after it becomes a clinical entity, it has a tendency to grow progressively worse. Whether or not it shall be checked in its course of advancement depends very much upon the intelligence with which the treatment is directed. All things else being equal, the man who understands tuberculosis best should be able to handle the case most successfully. In practice, however, it does not always work out in this way because we are not only dealing with a disease, but we are dealing with an individual; so, the prognosis in tuberculosis, in a given case depends not only upon the physician's knowledge of the disease, but upon his ability to successfully inspire the patient to a whole-hearted cooperation. The physician's responsibility in tuberculosis is greater than in almost any other ordinary disease because, at best, the disease is one which will extend over many months,—often several years,—and, during all this time it is the duty of the physician to guide the patient in such a manner that he is able to preserve his resisting power and keep his defensive forces acting to best advantage. The prognosis will usually depend upon the success with which the physician is able to accomplish this end.

Cooperation of the Patient.—No matter how intelligent the physician may be, and no matter how comprehensive and satisfactory his plan of treatment, unless the patient gives a whole-hearted cooperation his chances of recovery from active tuberculosis are not good. A patient may throw away all chances of recovery by some foolish act; or, he may turn an apparently hopeless prognosis into a favorable one by faithful cooperation.

Earliness of Diagnosis and Treatment.—The prognosis of tuberculosis depends largely upon the earliness of diagnosis and the time when proper treatment is instituted. The pathology of this disease is such that it becomes more complex as the disease advances. In the beginning, when the lesion is small, it is not such a difficult thing to prevent the disease from spreading, and keep the patient in the proper environment, both mental and physical, until his lesion is healed. While the lesion is small there is not the same opportunity for bacilli to escape and form metastases that there is later, for the foci are smaller and fewer in numbers than when the disease is extensive. Bacilli escape into adjacent tissues or into the blood stream during the periods when the foci of infection have become necrotic, caseate and break down. The greater the number of caseating tubercles, the greater the danger of such escape of tubercle bacilli, and the greater the demands upon the body to keep infection from occurring. Thus, it can be seen that a small lesion does not tax the organism's defensive powers nearly as much as a widespread lesion. No patient ever dies from the original focus, but through extensions and repeated extensions. It is to the prevention of these metastases, and secondary metastases that much of our therapeutic endeavor is directed. It is important to heal the lesion which already exists, and to that end we endeavor to raise the resisting power of the patient to the highest point possible, and to stimulate his foci of infection, by specific focal reactions, produced either by tubercle protein from his own tubercles, or by the tubercle protein introduced from without.

It is not difficult to see the great importance of treating the lesion while it is small. In this, tuberculosis is no exception to other diseases. It is far better to repair a small damage than to wait until it grows. This is a good rule in all lines of endeavor.

Early diagnosis, with proper treatment, in intelligent patients, should bring about an arrestment in at least 90 per cent of cases. After the disease has extended, however, and the early clinical tuberculosis has passed on into the moderately advanced stage, the prognosis has been reduced from 90 per cent to 50 or 60 per cent. The time required for this decline in prognosis to occur may be short or it may be long. Very often we see an early lesion change to one of moderately advanced activity within a period of a few weeks.

Time is the essential factor in tuberculosis. The time of the diagnosis, the time when treatment is begun, and the length of the time that the treatment is carried out are factors of the greatest importance.

Advanced tuberculosis, while not hopeless, as was formerly thought, proves to be very difficult to control. If we take the ordinary advanced cases of tuberculosis as they come to us, the chances of bringing about a healing are not very good. We can produce a healing, under ideal conditions, in about 10 per cent and an arrestment in about 30 per cent more, if we include both active and quiescent cases that come under our care. If we exercise care in choosing cases, and take only those which are more hopeful, we can bring about an arrestment in a very much larger percentage. On the other hand, if we confine our endeavors to those who are suffering from active advanced disease, with more or less constant toxemia, the percentage will be greatly reduced.

If the medical profession could only learn the importance of early diagnosis and the immediate treatment of tuberculosis, it would not take long to satisfy the most skeptical that tuberculosis is a curable disease even with our present indirect methods of treatment. If, on the other hand, we are to continue to spend our efforts trying to repair lungs which have been destroyed by the tuberculous process, and continue our attempts to restore the organism to a condition of efficiency, after this disease has become widespread, both laymen and physicians will continue to feel pessimistic regarding its prognosis.

Character of Treatment.—The prognosis depends very much upon the character of the treatment. Some patients get well without undergoing any definite course of treatment, even without knowing that they have had clinical tuberculosis. Most patients, however, when the active disease has made its appearance, require some form of active assistance if they are to recover their health. Tuberculosis requires a carefully planned therapy. This is particularly true because we have no definite, direct method of attacking it. Until we can find some specific remedy, like quinine in malaria, or salvarsan and mercury in syphilis, which will directly attack the tubercle bacillus and produce its destruction, we must work on in an indirect way. It is necessary at the present time, to deal largely with the patient's resisting power. While this is an indefinite and uncertain quantity, yet, with our recent advances in knowledge, it is becoming a more tangible asset. We must build up his nutrition; keep his metabolic activities at the highest point possible; aim to preserve his nervous equilibrium; a healthy gastrointestinal activity, and an efficient circulatory balance. This can only be done by carefully studying the patient as an individual and suiting the various remedies and measures which are at our command to his needs.

The only remedy we have, which has any specific action, is that derived from the tubercle bacillus itself. Preparations made from the bacillus are specific in that they produce protective substances against those portions of the bacillus contained within them and in that they react when they come in contact with the specific proteolytic enzymes which the body cells are stimulated to produce; and also in that such a reaction, if it occurs in and near the focus of infection, stimulates the production of fibrosis and healing.

The utilization of these remedial measures for the best interests of the patient is not easy. Many of them seem so simple that this fact alone defeats their proper employment.

I am a firm believer in the intensive treatment of tuberculosis. Success depends upon an intimate relationship between the patient and physician and their thorough cooperation. This association must continue over a prolonged period of time; and the various helpful measures must be adapted to the patient's particular needs as they change from time to time. Such treatment, when carried out with a whole-hearted cooperation on the part of the patient, makes the prognosis in tuberculosis quite favorable. It has been able to produce an arrestment in more than 90 per cent of early clinical tuberculosis; more than 60 per cent of moderately advanced cases; and more than 30 per cent of far advanced cases. The let-alone policy, which is carried out far too commonly, will reduce the percentages above mentioned, by 50 per cent. It is far more sensible to let a patient carry out a let-alone policy with typhoid fever, pneumonia, scarlet fever, and other acute infections, which run a short course, than it is to carry out such a policy with tuberculosis. The results would be no more disastrous than they have been, and still are, in tuberculosis, when such a course is followed. In this long drawn-out disease, which depends so much for favorable results upon a prolonged and hearty cooperation, close medical supervision with an intensive routine is an important factor in prognosis.

Sanatorium Versus Home Treatment.—When the diagnosis of tuberculosis is made the most important question to decide is whether the patient is to be treated in the home or in some sanatorium. From the standpoint of prognosis, there is no question as to which course should be pursued. From the standpoint of expediency, however, at times, it may be necessary to follow a course which offers less chances of cure. All else being equal, and granting that the patient is being treated by a physician with equal skill, whether it is in the home or in the institution, and that the institution is run in such a manner as to give the patient the benefits which such an institution should provide, there is no comparison in the two methods from the standpoint of prognosis.

A properly conducted sanatorium furnishes an ideal place for the application of the measures which are best and most useful in the treatment of tuberculosis. It removes the patient from the home with its cares and worries; relieves him from contact with business and its troublesome details; and places him in an atmosphere of helpfulness and hopefulness, where he has an opportunity to associate intimately with trained physicians and attendants, all of which has a tendency to improve his chances of cure.

In the home, on the other hand, the difficulties are many, and oftentimes unsurmountable. Home cares; home worries; a failure to keep up the proper program; the attempt to make the home, which was intended for the well, a place for the sick, while it, at the same time, is being inhabited by those who are well; and the association with those who are interested and engaged in the usual activities of life, make it almost impossible for the patient to keep up a cooperation sufficiently long for the disease to become arrested, or healed. When I have placed my patient in the sanatorium, I feel that his chances of recovery have increased from 25 to 50 per cent (See Volume II, Chapters XLVII and XLVIII.)

Open Air.—Open air as a curative measure in tuberculosis is universally recognized, and rightly so; because there is no other single factor, which is so generally applicable and which is capable of giving the patient equal aid. Patients can get well of tuberculosis without being treated in the open; but, when we realize that the great value in the air is the physical effect which it exerts upon the body, the value of treating a patient in the open air, as compared with treating him in rooms, particularly in closed rooms, is self-evident.

It is desirable that those who are treating tuberculosis should know the limitations of open air therapy, and realize that it is only one of many measures for the improvement of the patient's general resisting power; at the same time, they should fully realize that its intelligent application materially increases the patient's chances of cure. When weather and climatic conditions are favorable, open air should always be employed. While it is possible to treat patients under conditions where the full benefit of open air cannot be satisfactorily obtained, it is all the more necessary under such circumstances to bring to the aid of the patient other measures which influence metabolism, such as psychotherapy, hydrotherapy, good food, and rest. Open air, as I have described more fully in Volume II, Chapter XXXVI, improves the nutrition of the skin, stimulates the superficial nerve endings, and, through them, influences the cellular activity of the internal viscera. The reaction which ensues facilitates gaseous exchanges and favors the maintenance of heat equilibrium; thus producing a beneficial influence upon the entire metabolic activity of the organism.

Climate.—Not many years ago climate was considered to be the main factor in the treatment of tuberculosis. It was thought that if a patient could live under certain climatic influences, that his chances of cure would be good; and, further, that if he could not obtain such climatic change, little could be expected.

For a time it was thought that these favorable climatic conditions were found only in the high mountains. This idea was based on an article published in the Edinburgh Journal in 1848 by Archibald Smith, who visited the Andes and noted that, while tuberculosis was extremely common at Quito, there was very little in the mountains above this city; consequently, he came to the conclusion that there was an immune zone somewhere between the altitude of Quito and the small cities of the mountains. This observation was confirmed in other high mountain resorts. It was seen that while there was much tuberculosis in the large cities of Germany and France, there was little in the high altitudes; while there was much tuberculosis in our eastern cities, there was little in the Rockies; consequently, it was accepted as a fact that treatment at high altitude offered the best chances of cure.

Further observation, shows that there is no zone immune to tuberculosis. Tuberculosis exists in all sections of the globe, and under all climatic conditions. It is most prevalent where people mass together, being particularly common in cities. It is an economic and social disease. In the sparsely settled districts, whether they be at an elevation, or at sea level, or even below sea level, as in our own Salton Desert, and the Steppes of Russia, there is very little tuberculosis. Tuberculosis can, and will, develop anywhere. People are infected in childhood. They carry the bacilli in their tissues as they grow older; and, if conditions are brought about, which tend to lower their resisting power, the bacilli may spread, multiply, and produce clinical tuberculosis. Such alterations in defensive powers are less apt to occur where hygienic conditions are maintained and where people live lives conducive to strength, as they do in country districts. We must not forget the influences exerted upon the body metabolism by sunlight and humidity and other physical principles. There is no question that conditions more favorable to metabolism are found in climates which favor an out-of-door life, which have a large percentage of sunshine and low humidity, as compared with areas where dampness and cloud prevail. A country which invites the patient to be out of doors, and which offers physical surroundings in the way of natural scenery and vegetation which are pleasing to the physical sense, will undoubtedly improve the patient's chances of recovery (see Volume II, Chapter XXXVII).

Heliotherapy.—Heliotherapy adds to the patient's chances of cure. In superficial lesions, particularly those accompanied by ulceration, the curative influence of sunlight is quite marvelous. Many ulcerations which, formerly, proved very difficult to handle, by long tried surgical methods, have yielded quite readily to modern sun and air treatment. While these may be aided to a certain extent by exposure to the air, exposure to sunlight seems to have an added curative influence.

The effect of sunlight, however, is not confined to its influence on local lesions. The influence which it has upon metabolism, through its stimulating effect upon the surface of the body, is an important factor. We must not lose sight of the fact that a very important effect in heliotherapy may be its influence upon the blood which circulates through the superficial capillaries. Heliotherapy should be employed as an important physical measure in building up the patient and improving his general resisting power. Unfortunately, it can be used only to a limited extent in some climates; but, wherever the sun shines, heliotherapy can be utilized to some extent, although not to its fullest advantage (see Volume II, Chapter XLIII).

Hydrotherapy.—This is a measure which can be used in any climate and adapted to all conditions, providing the physician understands the physiological effects of water. I have never seen a patient to whom baths could not be advantageously adjusted. Much of the failure in the application of hydrotherapy has been due to the fact that the use of water seems so simple. When one realizes, however, that the condition of the patient, the presence or absence of toxemia, the condition of his superficial blood vessels, the state of his metabolic activity, the condition of the atmosphere which surrounds him, the temperature of the water, the mechanical impact of the same, and the use or non-use of friction during and after its application, are all variables which enter into the effect produced, one can see that attention to detail in its application is absolutely essential to success. When adapted to the needs of the individual, however, we have in hydrotherapy a very useful measure for improving the patient's metabolic activity and increasing his power to overcome disease (see Chapter XLII).

Food.—The prognosis in tuberculosis depends upon a sufficient dietary. No matter what other measures are employed, a sufficient amount of food must be used by the patient if he is to regain health. While he may go through periods when the amount of food eaten is not sufficient to supply the demands of his body, yet, if these are long continued, malnutrition will follow, which greatly interferes with the patient's chances of cure.

There has been much discussion about what constitutes an adequate dietary. There is no unanimity of opinion as to what dietary is the most useful for the tuberculous patient; at the same time, all agree that the patient should be supplied with a sufficient number of calories to take care of his ordinary metabolic changes, plus the extra demands which are made upon him by his disease, and still have a surplus for increasing his defensive powers. Some observers favor a diet rich in fats; others rich in protein; and, occasionally, we find someone who believes that extra large amounts of carbohydrates should be employed.

Suffice it to say, that our digestive capabilities are such that when reinforced by the metabolic activities of the cells, we are able to derive sufficient nutriment from dietaries which are not formed according to our scientific ideas. A patient can nourish himself on foods which are largely carbohydrate, largely protein, or largely fat; at the same time, it is undoubtedly wise to supply the body with foods belonging to each of these classes in quantities which experiments have shown to be the most rational.

From the prognostic standpoint, nutrition is the goal to be aimed at, not fat. Nothing is gained, however, by putting on weight if it is necessary, in order to do so, to give the patient large quantities of food over a prolonged period of time. Overfeeding is still too commonly found in the treatment of this disease. The patient is led to believe that if only he can keep fat he will get well. Fat is, prognostically, bad rather than good. The fat patient is soft, not resistant. He has too much extra tissue to care for, without having a corresponding increase in strength and vigor (see Volume II, Chapter XXXIX).

Rest and Exercise.—The prognostic influence of rest is recognized by all students of phthisiotherapy. In the writer's personal experience, he has gradually learned to have great respect for the influence of these two measures when properly applied. I am sure, as I look back on some of the results in my early practice, that many failures could have been prevented had I fully realized the importance of rest in treatment. A patient's chances of recovery are increased greatly if he is confined to bed during the entire period of not only clinical activity, as expressed in the symptoms which accompany toxemia, but also during the period when there are widespread lesions in the lung, accompanied by signs and symptoms, which indicate that the pathological changes are still taking place rapidly. I am inclined now to keep my patients at rest until I feel reasonably certain that there is no further danger of necrosis and caseation taking place. While it is difficult to assign to the various measures which we employ their true part in the production of a favorable result, yet I am sure that I cannot emphasize too strongly the prognostic importance of rest during the early months of treatment of all patients who are suffering from tuberculosis.

Exercise is likewise a valuable adjunct and adds greatly to the chances of cure by improving the patient's metabolism and making him strong and more resistant. It does this, however, only when it is nicely adjusted to the individual patient's particular pathology. If one is to err in prescribing either rest or exercise, let it be on the side of rest (Volume II, Chapter XXXVIII).

Psychotherapy.—As mentioned elsewhere in this monograph, one of the factors which is capable of doing most good in the treatment of tuberculosis is psychotherapy. If employed to its fullest value, it can even make up some of the deficiencies which come from a failure to fully utilize other measures. I have often said that I would rather have a patient treated in a room, if I could give him the benefit of hydrotherapy and psychotherapy, with good food, than treat him in the open air, with hydrotherapy and psychotherapy omitted. The contentment and cooperation of the patient is such an important factor that I cannot emphasize too strongly the importance of this measure in the treatment of tuberculosis. The effect of this measure is illustrated by an incident which came under the writer's notice recently. Two physicians were treating the same group of patients,—one of whom had a strong personality and used psychotherapy to its full value, while the other lacked magnetism and was more or less mechanical in his methods. A patient was going to the office for treatment and was sometimes treated by one and sometimes by the other. One day the patient remarked to the physician first described: "I do not know why it is; you and your associate use the same measures, and both give tuberculin in the same manner, but when I leave your associate I feel that I have had nothing but the injection. When I leave you I feel happy, contented, and relieved of all apprehension for the day." This experience is not an uncommon one. Personality is a great factor in medicine. All physicians who are desiring to be successful in the treatment of a chronic disease like tuberculosis should cultivate cheerfulness, optimism, friendliness and sympathy, that they may give their patients not only the benefit of the usual physical measures, but also the full physiological benefit that comes from a helpful psychic attitude (see Volume II, Chapter XLI).

Tuberculin.—Of all the remedies used in tuberculosis, tuberculin is the only one that has a direct specific influence upon the tuberculous tissue. Tuberculin is employed in the treatment of tuberculosis for two distinct purposes; one, that of producing immunity against the products of the tubercle bacillus; the other, that of producing a focal reaction which stimulates the production of fibrosis.

It was formerly thought that the production of immunity was the chief function of tuberculin; but, as we study its action more and more, this seems to be open to question. There is no doubt that any foreign protein will produce an immunity against itself, so that the various components of the tubercle bacillus, which are found in the individual preparation of tuberculin employed must,, of necessity, establish a resistance on the part of the body cells against themselves; and there is further no doubt that this has considerable influence in keeping up a specific resistance against the tubercle bacillus; but in most cases of tuberculosis, resistance to the bacillus is already high, and an increased immunity is not so important. The patient constantly gives himself doses of bacillary products from his own focus; and, in this way, gets the identical stimulation which is essential to the production of a full immunity against the bacillus. As far as we know, we are not able to obtain this regularly by any preparation of tuberculin that we have hitherto employed.

Any specific product made from the tubercle bacillus, however, possesses the peculiar properties of producing a reaction between itself or the products which result from its chemical decomposition, and the body cells. When this reaction occurs in, and near the focus of infection, it produces a stimulation, which results in hyperemia or congestion, which hastens healing.

When tuberculin is intelligently employed it favorably influences prognosis. It is as difficult to estimate fully the value of tuberculin in the treatment of tuberculosis as it is to estimate the value of fresh air, good food, psychotherapy, and climatic conditions; because when tuberculin is used, many of the other measures are likewise employed. After twenty years' experience in the use of tuberculin, I have no hesitancy in saying that this remedy, when properly suited to the individual patient under treatment, and not given haphazardly, or by routine, will add at least a minimum of 20 per cent to the chances of healing.

I further believe that, if carried out sufficiently long the tuberculin treatment will unquestionably produce a firmer scar and more complete healing. It is my observation that those patients who have been successfully treated with tuberculin and in whom the treatment has been kept up for a sufficient length of time, show less tendency to relapse than those who are treated by the usual dietetic, hygienic measures alone.

Tuberculin is not a remedy that can be used to best advantage by men who are unaccustomed to its use; consequently, its favorable influence will have to be denied to a large percentage of those who are suffering from this disease. The value of this remedy has been recognized for a long time by those who are ordinarily opposed to its general use, in those cases which have failed to make satisfactory progress without its employment. They justify its use in such cases by saying that it seems to furnish the stimulation necessary to start these cases, which have hitherto failed to respond to ordinary measures, on the road to recovery. If of value in these cases, it stands to reason that it must be of value in others, although its influence might not be so apparent in those which show a general tendency toward healing without it. When one bears in mind, however, that any healing that occurs in tuberculosis takes place because of the specific focal stimulation produced by the products of the tubercle bacillus, the rationale of this remedy is evident (see Volume II, Chapter XL).

Induced Pneumothorax.—During the past decade induced pneumothorax has been gradually gaining supporters among those who are interested in the cure of tuberculosis. The theory on which its employment rests is that it puts the diseased organ at rest; compresses the areas of activity and necrosis; prevents the absorption of toxins; and, by so doing, relieves the patient of the debilitating action of toxins and enables him the better to build up his defensive powers. Induced pneumothorax is supposed to be applicable only to one-sided lesions, although it has been tried by some during more recent times, where both lungs were involved, compressing one side at a time.

That pulmonary compression at times aids in the cure of tuberculosis cannot be denied, and there is no doubt in my mind but that many men will give their patients better chances of cure by putting the lung at rest, and filling the pleural sac with gas than they would by not doing so. On the other hand, the percentage of cases where it is essential to cure, from my observation, are very few. The cure of tuberculosis, even when induced pneumothorax is used, does not consist alone in filling the pleural sac with gas. This must be kept filled, and the patient must be cared for and treated the same as when this is not used. Its final effects must also be considered. There are a certain number of accidents which attend its employment, such as pleural shock, rupture of a cavity wall, pyopneumothorax, and causing the disease to become more active in the other lung. The after-effects must also be considered. From the physiological standpoint it is impossible to compress the entire lung over a prolonged period of time, without producing a severe strain upon the heart. There is considerable question whether at the end of the period of a few months or a year or more, that is necessary to produce a result by this measure, the heart will not have been subjected to a strain which will eventually prove to be the patient's undoing. The ordinary hygienic treatment, with proper psychotherapeutic influences and tuberculin added, under ideal conditions, is probably more satisfactory and more rational in the handling of the disease than is induced pneumothorax; nevertheless, there are many patients who for one reason or another, cannot be given these other measures to best advantage for whom induced pneumothorax, with other helpful measures that can be employed, will offer better chances of cure than though it were omitted. In estimating the value of this measure one must bear in mind that it is good lung tissue which collapses first; consequently, if full value is to be obtained from its employment, the compression must be complete (see Volume II, Chapter XLIV.)

Pharmacological Remedies.—Pharmacological remedies, now and then, have a very important influence in the treatment of tuberculosis. There are certain remedies which are of great value in improving the symptoms and complications which exist, and without which it would be very difficult to treat the patient successfully. Among such may be mentioned the various forms of opium used for pain and cough; atropin to combat many of the vagotonic conditions; hydrochloric acid, in cases of deficient gastric secretion; laxatives which, of course, should be reduced to the minimum, the condition of the bowels being controlled as largely as possible by dietary and other measures; sedatives, other than opiates, such as the bromides, for the relief of nervousness; remedies for sleep, when this cannot be induced by other measures; and last, but not least, we must not forget such preparations as digitalis, strychnine, iron and arsenic, which can often be used to the advantage of the patient.

In speaking of the prognostic value of pharmacological remedies we must bear in mind that the relief of troublesome symptoms is often of inestimable value to the patient. Some symptoms, such as pain and sleeplessness, while, in themselves, not so dangerous, produce certain impressions upon the nerve cells which result in depression and general interference with cellular activity; so, we cannot ignore the importance of these common symptoms in their effect upon the prognosis in tuberculosis. We can favorably influence prognosis by relieving nagging, distressing symptoms and complications; so, we must not neglect the little things that add comfort and make for contentment and happiness during the long period necessary for cure (see Volume II, Chapter XLVI).

Change of Occupation.—Much foolish advice is often given to patients suffering from tuberculosis, or who have recently obtained an arrestment of their disease. Probably no advice is given more readily than that of telling the patient to change his occupation,—to get out in the open air. Such advice should not be given so readily; and during recent years, instead of advising my patients to change their occupation, and get out in the country, I have been looking upon the matter from what I believe is a more sensible standpoint. The occupation followed by the patient, as a rule, occupies about one-third of his time. It is not so much what the patient does during the one-third of the time as it is the other two-thirds of his time that determines whether or not he is to regain his health.

Oftentimes patients are told to go to the country and take up some new work, for which they are totally unfitted by nature, and are often unable to do financially. The result is that the patient obtains the fresh air but is compelled to learn a new occupation and to make financial sacrifices which greatly reduce his comfort, and interfere with his state of health. It is more sensible when a patient consults a physician as to his occupation, to find out whether or not he does his work easily, whether he knows the work that he has been doing; and if he does it easily, tell him to continue it, but to look well to the other two-thirds of the twenty-four hours which remain after his work is done. If he will give himself proper rest, see that he has substantial food, sleep out of doors, and avoid excess, the eight hours of work in the office, or mill, or shop, as a rule, will be no more harmful to him than undertaking to master a new work, or new profession for which he has no particular aptitude and which causes an entire rearrangement of his life and habits, even though the latter is in the open. This fact is better understood now that we know that it is not the lack of oxygen in the inside air that proves harmful (see Volume II, Chapter XXXVI).

APPENDIX

Owing to an unavoidable delay, considerable time has elapsed between the printing of the main portion of the second edition of this book and its completion. In the meantime I have been able to amplify my classification and grouping of symptoms as given in Volume I, page 366, and desire to publish them as an appended chapter.

SYMPTOMS OF PULMONARY TUBERCULOSIS

GROUP I

SYMPTOMS DUE TO TOXEMIA

Caused by Harmful Stimulation of	*Symptoms*
I Nervous System in General	1. Malaise
	2. Lack of endurance
II Endocrin System in General	3. Loss of strength
	4 Nerve instability.
III Sympathetic Nervous System	5. Diminished digestive activity
IV Sympathicotropic Endocrins particularly adrenals and thyroid.	6. Increased metabolic rate.
	7. Loss of weight
	8. Increased pulse rate
	9. Night sweats
	10. Temperature
	11. Leucocytosis

It will be seen that those symptoms in Group I, designated as the group caused by toxins, can be divided into two subsidiary groups. The first four symptoms: malaise, lack of endurance, loss of strength and nerve irritability are not characteristic of toxic action alone, but may result when a patient is tired out, worried, or nervously exhausted as well. This group of symptoms results from harmful stimuli which act upon the general controls of the body tissues, of the nervous and endocrin systems as a whole. The remaining symptoms of this group: digestive disturbances (hypomotility and hyposecretion), metabolic disturbances resulting in the loss of weight, increased pulse rate, night sweats, temperature, and blood changes, are more specific in their etiology. These are characteristic of harmful stimulation of the sympathetic component of the vegetative nervous system and of harmful stimulation of those glands of internal secretion which are sympathicotropic in nature, of which the adrenals and thyroid are the best examples.

SYMPTOMS OF PULMONARY TUBERCULOSIS

GROUP II

REFLEX SYMPTOMS FROM THE LUNG

Inflammation of Lung

Afferent Nerves	*Symptoms*	*Efferent Nerves*
Afferent through (vagus) parasympathetics	Hoarseness	Laryngeal nerves.
	Laryngeal irritation . . .	Superior laryngeal nerve.
	Cough	Laryngeal and nerves to all expiratory muscles with inhibition of nerves to inspiratory muscles.
	Inhibition of heart . . .	Motor fibers of cardiac vagus.
	Increased muscle tonus and glandular secretion in gastrointestinal canal .	Motor fibers of gastric and intestinal parasympathetics.
	Flushing of face	Sensory fibers of trigeminus.
	Spasm of sternocleidomastoideus and trapezius .	Accessorius.
	Deviation of tongue from median line.	Hypoglossus.
	Degeneration of facial muscles	Trigeminus.
Afferent through sympathetics	Flushing of ear	Third sensory cervical.
	Dilatation of pupil . . .	Motor from Budge's Center (lower cervical and upper dorsal).
	Spasm of muscles of shoulder girdle and diaphragm	Cervical motor nerves, IInd to VIIIth.
	Lessened motion of chest wall, partly due to muscle spasm as above . .	Cervical motor nerves, IInd to VIIIth.
	Pain above 2nd rib and spine of scapulae (superficial)	Cervical sensory nerves, particularly IIIrd, IVth, and Vth.
	Pain in muscles of shoulder girdle (deep pain) . .	Cervical sensory nerves, IInd to VIIIth.
	Degeneration of skin and subcutaneous tissue above 2nd rib anteriorly and spine of scapulae .	Cervical sensory nerves, IIIrd, IVth, and Vth.
	Degeneration of muscles of shoulder girdle	Cervical sensory and motor, IInd to VIIIth.

In Group II, which includes the reflex symptoms, I have made particular amplification. In this group, I have discarded all distinction between symptoms and signs, and between subjective and objective symptoms and included not only the common reflex symptoms but some that are found more rarely. I have arranged these in such a manner that one can see at a glance the path through which the reflex is produced. The afferent nerve appears in column one, the symptoms in column two and the efferent nerve which completes the reflex action in column three.

SYMPTOMS OF PULMONARY TUBERCULOSIS

GROUP III

SYMPTOMS DUE TO THE PROCESS *per se*

Spitting of blood
Sputum
Frequent and protracted colds (tuberculous bronchitis)
Pleurisy (tuberculosis of the pleura).

In Group III, which includes those symptoms which are more particularly due to the local action of the tuberculous process we have two symptoms and two syndromes. Hemorrhage, due to rupture of a vessel wall or an inflammatory process accompanied by a sanguineous discharge, and that form of sputum which results from a rupture of tubercles, are symptoms of tuberculosis, which are due to the local action of the disease process. Frequent and protracted "colds," and pleurisy, on the other hand, are really syndromes expressive of active tuberculosis, one in the pulmonary tissue, the other in the pleura; and as such are accompanied by symptoms of Groups I and II. Hemorrhage and Sputum may or may not be accompanied by symptoms of Groups I and II.

The most important fact in this classification is not that an explanation is offered for each symptom, although that is a great advance in our knowledge of the disease; but the fact that there are only three factors acting, although they express themselves in as many ways as there are symptoms. We should now approach the analysis of the symptoms of active tuberculosis with these questions: are there any toxic symptoms present? are there any reflex symptoms present? are there any symptoms which may be attributed to the local action of a tuberculous process? We must not expect to find all symptoms of each group present in every case, but it suffices to find some.

For further discussion of these groups the reader is referred to the treatment of the subject in the main portion of the text.

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